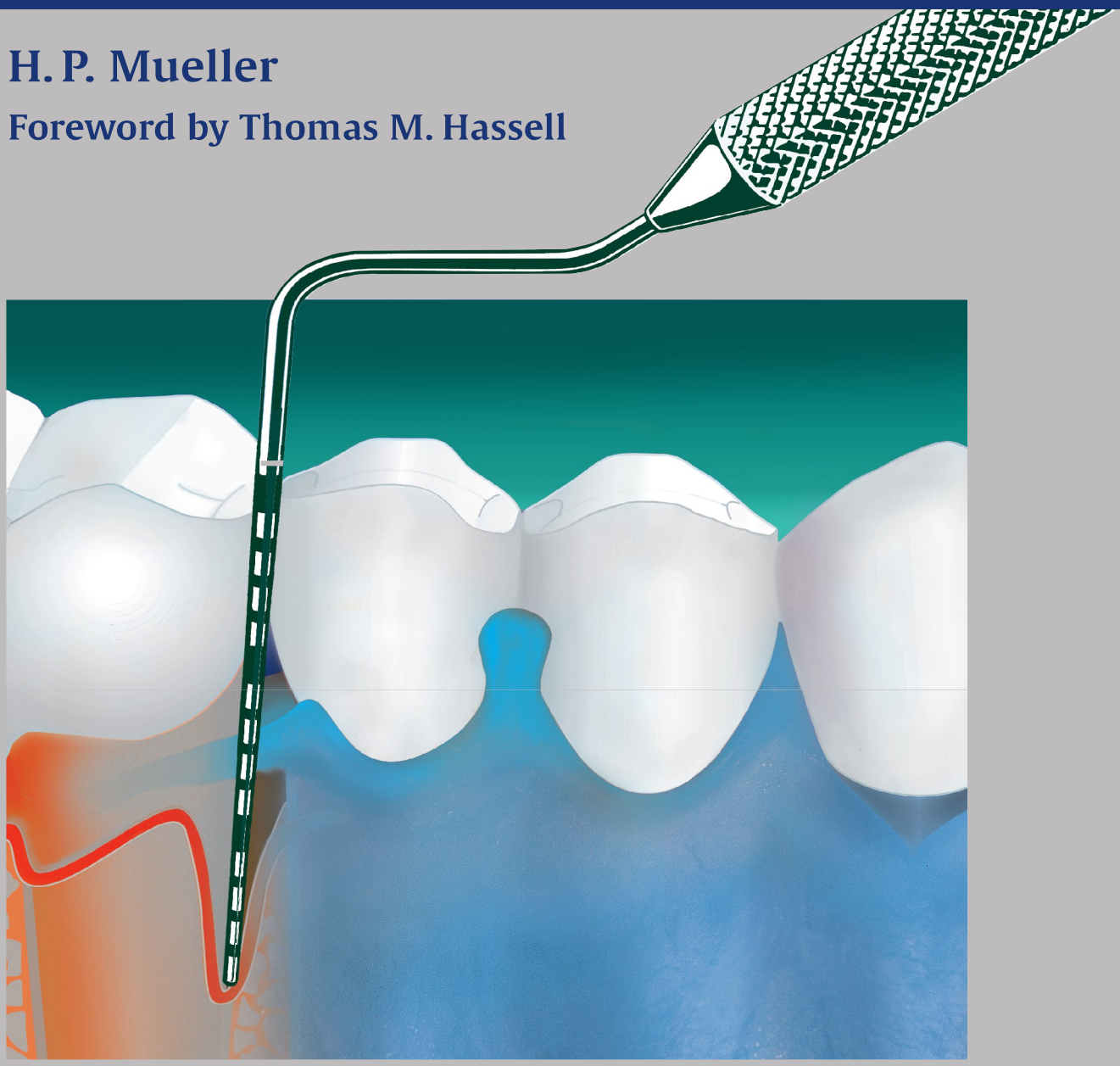
Periodontology

The Essentials

H. P. Mueller

Foreword by Thomas M. Hassell



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Periodontology

The Essentials

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241 illustrations
33 tables

Thieme
Stuttgart · New York

Library of Congress Cataloging-in-Publication Data

Müller, Hans-Peter, Prof. Dr. med. dent.
[Parodontologie. English]
Periodontology : the essentials / Hans-Peter Müller.
p. ; cm.
Includes bibliographical references.
ISBN 1-58890-355-9 (alk. paper) –
ISBN 3-13-138371-2 (alk. paper)
1. Periodontal disease-Handbooks, manuals, etc.
2. Periodontics-Handbooks, manuals, etc.
[DNLM: 1. Periodontal Diseases-Handbooks. WU 49 M954p 2004a] I. Title.

RK361.M8413 2004
617.6'32–dc22

2004017259

© 2005 Georg Thieme Verlag,
Rüdigerstrasse 14, 70469 Stuttgart, Germany
http://www.thieme.de
Thieme New York, 333 Seventh Avenue,
New York, NY 10001 USA
http://www.thieme.com

Typesetting by Satzpunkt Ewert GmbH, Bayreuth
Printed in Germany by Druckhaus Götz, Ludwigsburg
ISBN 3-13-138371-2 (GTV)
ISBN 1-58890-355-9 (TNY) 1 2 3 4 5

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Foreword

The past several years have witnessed the publication of numerous "classic" textbooks of periodontics and periodontology by well-known scholars and acknowledged scientists. These are large and weighty and expensive tomes, with thousands of literature citations and hundreds of pages of scientific minutia. The question that remains, however, is the value of such huge works in the day-to-day dental or periodontal practice, or for a student who needs a fast reference and a solid, no-nonsense but up-to-date answer to a practical clinical question. This latter is the niche that is now filled by *Periodontology – The Essentials*, by Dr. Hans-Peter Mueller. It is a welcome and timely new book, and will certainly be appreciated by students of dentistry and dental hygiene, private practitioners of general dentistry, and periodontists as well.

This compact yet comprehensive work clearly portrays the critically important paradigm shift that has occurred in dentistry and particularly periodontology in the past decade, a shift away from reparative and sporadic treatment of varying dental and periodontal clinical symptoms and toward a comprehensive and almost holistic approach to total patient care. The book embraces a newly defined goal, based upon the recent renaissance of an ages old concept: Focal infection as an etiologic factor in systemic diseases. The now-acknowledged fact that periodontal disease enormously increases each patient's risk of serious medical consequences, including even untimely death, has changed forever the profession's approach to inflammatory oral disorders. For decades, oral health care professionals have attempted to "motivate" their patients toward excellent oral hygiene, but seldom with success of long duration, because the *consequences of non-compliance* were not life-threatening or otherwise of particular importance. No more.

Today, the scenario has changed dramatically, and the demands of our patients have increased enormously. Patients present today with a panoply of demands: immediate and long-lasting oral/dental health and function, dental esthetics above all, and *prevention* of systemically-related consequences such as cardiovascular events and more.

Dr. Mueller's book addresses these new and patient-centered exigencies directly and adroitly, presenting the traditional topics of Phase 1 Therapy and (subsequent) surgical intervention in a new and refreshing light. This book addresses the *consequences* of periodontal treatment as well as the technical aspects of daily patient care.

The author has invested extensive time and effort to freshly describe the etiology and pathogenesis of various disorders of the tooth-supporting structures, encompassing the most contemporary clinical and basic research findings. The literature citations are up-to-date but limited to the most significant articles that relate to daily practice. Only thorough knowledge of the cause(s) of the inflammatory disease processes will lead to effective choices in treatment planning and therapy. Included in the book are the most recent findings concerning the *hereditary* basis for periodontal diseases, including the now-acknowledged "un-alterable" genetic risk factors, and strategies for approaching these most-difficult clinical syndromes.

Some of the most contemporary research into disease pathogenesis (pro-inflammatory mediators etc.) as well as a modicum of "futurology" in terms of how the profession will move, likely rapidly, into the use of, for example, growth factors, in periodontal therapy are covered in an easy-to-understand manner, and with the caution that is due even today until such advanced treatment modalities have stood the test of time.

In sum, Dr. Mueller's new book presents a refreshing new look at the old profession of "gum gardening." The book provides astonishingly complete coverage of a rapidly-advancing medical/dental discipline, with a minimum of verbiage to confuse the practitioner or obfuscate the basics of the issue at hand. The illustrations are of high quality and reduced to the basics required for understanding a concept. Color illustrations have been kept to a minimum, but color

is used effectively in important conceptual circumstances.

I am pleased and proud to introduce this new book, which effectively bridges the gap between scientific esoterica and the practitioner's daily need for relevant knowledge.

Flagstaff, Arizona, USA, in August, 2004
Thomas M. Hassell, DDS, PhD

Preface

In the realm of Dentistry, the discipline of Periodontology has seen in the past 10 years a remarkable independent development: basic and clinical research converges, and progress in Periodontology accelerates exponentially[1]. Periodontal research is nowadays done in teams, in which clinicians work hand in hand with biologists, behavioral scientists, epidemiologists, and specialists of different other medical disciplines. Moreover, very recently, workshops have been organized by the European and American Academies of Periodontology which addressed questions of paramount importance for the clinician by identifying and evaluating the current literature. These workshops resulted in a number of systematic reviews, which are regarded the highest level of evidence.

Impressive advancement of our knowledge has to be implemented in our teaching as soon as possible. This may be one of the most apparent advantages of the present compendium on essentials in contemporary Periodontology. Unlike traditional and sometimes even classic textbooks it may offer, in a very condensed form, the latest developments in the field. It should be kept in mind, however, that a compendium cannot and should not replace diligent reading textbooks on Periodontology and original literature. Although undergraduates are the main target for the compendium, and extensive reference lists are therefore not provided, also general practitioners and specialists in other fields of Dentistry may take the opportunity, too, of quickly checking specific periodontal details in their daily practice.

I gratefully acknowledge critical comments of several colleagues I had the privilege to work with in the past years. It is a deep desire to thank Drs. Eino Honkala, Mauno Könönen, Eero Kerosuo, Jawad Behbehani, and Bobby K. Joseph at Kuwait University, for fruitful exchange of ideas and development of teaching conceptions. I am especially grateful to Dr. Eija Könönen, who thoroughly reviewed the chapter on Periodontal Microbiology. I wish that this Compendium might serve as a valuable supplement to clinical teaching in Periodontology.

Kuwait City, August 2004

Hans-Peter Müller, DDS, Dr. med. dent., PhD

[1] Page RC. Milestones in periodontal research and the remaining critical issues. J Periodont Res 1999; 34: 331–339.

Contents

Background

1 Anatomy and Physiology 2
Development 2
General Remarks 2
Crown Development 3
Root Development 3
Cementogenesis 3
Marginal Periodontium 5
Macroscopic and Microscopic Anatomy 5
Oral Mucosa 5
Gingiva 6
Root Cementum 7
Periodontal Ligament 8
Alveolar Bone Proper 9
Physiology 9
General Remarks 9
Turnover Rates 9
Defense Mechanisms 10
Possibilities of Repair 10

2 Periodontal Microbiology 12
Ecology of the Mouth 12
The Oral Cavity as a Biotope 12
Colonization Mechanisms 12
Biofilm Dental Plaque 13
Formation of Supragingival Plaque 14
Colonization of the Subgingival Region . 15
Dental Calculus 16
Periodontitis: An Infectious Disease 16
Identification of Periodontal Pathogens . 16
Types of Infection 17
The Susceptible Host 19
Presence of Pathogens 19
Absence of Beneficial Microorganisms .. 20
Conducive Environment in the Periodontal Pocket 21
Transmission 22

3 Pathogenesis of Plaque-Induced Periodontal Diseases 24
Pathogenesis of Plaque-Induced Gingivitis 24
General Remarks 24
Initial Gingivitis 25
Early Gingivitis 29
Established Gingivitis 29

Pathogenesis of Periodontitis 31
Advanced Lesion 31
Formal Pathogenesis—Progression 34
Characteristics of a Multifactorial Disease 36

4 Epidemiology of Periodontal Diseases . 38
Epidemiological Terminology 38
General Remarks 38
Periodontal Epidemiology 38
Examination Methods 39
General Remarks 39
Assessment of Gingival Inflammation .. 39
Bacterial Deposits 40
Combined Indices 40
Attachment Loss 40
Epidemiology of Plaque-Induced Periodontal Diseases 41
General Remarks 41
Natural History 42
NHANES III 43
Europe 45
Early-Onset (Aggressive) Periodontitis . 45
Prevalence, Extent, and Severity of Gingival Recession 46

5 Prevention of Periodontal Disease 47
Prevention-Oriented Dentistry 47
General Remarks 47
Karlstad Study 47
Possibilities for Prevention 48
Measures at the Population Level 48
Secondary and Tertiary Prevention 49
Preferential Treatment of High-Risk Groups 49

6 Classification of Periodontal Diseases . 50
Current Classification 50
General Remarks 50
Plaque-Induced Gingival Diseases 52
General Remarks 52

Systemically Modified Gingival Diseases 52
Drug-Influenced Gingival Diseases 52
Gingival Diseases Modified by Malnutrition 53
Gingival Diseases of Specific Bacterial Origin 53
Gingival Diseases of Viral Origin 53
Gingival Diseases of Fungal Origin 53
Gingival Manifestations of Mucocutaneous Diseases 54
Allergic Reactions 55
Traumatic Lesions 55
Foreign Body Reactions 55
Periodontitis 56
Chronic Periodontitis 56
Aggressive Periodontitis 56
Periodontitis as Manifestation of Systemic Disease 56
Necrotizing Periodontal Diseases 57
General Remarks 57
Necrotizing Ulcerative Gingivitis 58
Necrotizing Ulcerative Periodontitis 58
Abscesses of the Periodontium 58
Gingival Abscess 58
Periodontal Abscess 58
Pericoronal Abscess 59
Combined Periodontic-Endodontic Lesions 59
Developmental or Acquired Deformities and Conditions 59
Localized, Tooth-Related Factors That Modify or Promote Plaque-Induced Gingival Diseases/Periodontitis 59
Mucogingival Deformities and Conditions Around Teeth and on Edentulous Alveolar Ridges 59
Occlusal Trauma 59

Diagnosis

7 Diagnosis of Periodontal Disease 62
History 62
General Remarks 62
Medical History 62
Dental History 62
Clinical Examination 63
Extraoral Examination 63
Intraoral Examination 63
Functional Examination 65
Periodontal Examination 65
Mucogingival Examination 69
Oral Hygiene 71
Radiological Examination 72
General Remarks 72
Panoramic Radiographs 72
Full-Month-Radigraphic-Survey 72
Computed Tomography 75
Intraoperative Diagnosis of Defect Morphology 75
General Remarks 75
Osseous Defects 75
Furcation Involvement 76
Advanced Diagnostic Techniques 76
General Remarks 76
Diagnostic Test Systems 76
Microbiological Tests 79
Markers of Specific and Nonspecific Host Response 83
Human Genetic Tests 85
Bad Breath 86
Diagnosis 86
General Diagnosis 86
Tooth-Related Diagnosis 87
Prognosis 88
Treatment Planning 88
General Remarks 88
Case Presentation 88

8 General Medical Considerations 89
Systemic Phase 89
General Remarks 89
Infectious Patients 89
Increased Risk of Infective Endocarditis 89
Further Indications for Antibiotic Prophylaxis 91
Bleeding Disorders, Anticoagulant Therapy 91
Atherosclerosis, Cardiovascular Diseases 92
Associations Between Marginal Periodontitis and Cardiovascular Diseases 92
Diabetes Mellitus 93
Pregnancy 95
Osteopenia, Osteoporosis 96
Smoking 96

Therapy

9 Emergency Treatment 100
Periodontal Emergencies 100
Traumatic Insults 100
Necrotizing Ulcerative Periodontal Disease 100
Herpetic Gingivostomatitis 101
Periodontal Abscess 101
Combined Periodontic–Endodontic Lesions 102

10 Phase I: Cause-Related Treatment 103
Mechanical Plaque Control 103
General Remarks 103
Tooth Brushing Techniques 103
Interdental Hygiene 104
Chemical Plaque Control 105
General Remarks 105
Chlorhexidine 107
Triclosan 107
Metal Salts 107
Other Additives 108
Local Anesthesia 108
General Remarks 108
Adverse Effects of Local Anesthetics ... 108
Regional Infiltration and Block Anesthesia 109
Supragingival and Subgingival Scaling and Root Planing, Subgingival Curettage 109
General Remarks 109
Definitions 109
Aims 109
Indication 109
Contraindication 109
Instruments 110
Procedure 112
Critical Assessment 114
One-Stae, Full-Mouth Disinfection 116
Reevaluation 116
Sharpening of Instruments 116
General Remarks 116
Sickle Scalers 116
Universal Curettes 117
Area-Specific Curettes 117

11 Phase II: Corrective Procedures 119
Periodontal Surgery 119
General Remarks 119
Gingivectomy 119
General Remarks 119
Aims 120
Indications 120
Contraindications 120
Instruments 120
Procedure 121
Postoperative Care 122
Critical Assessment 122
Gingivoplasty 123
Definition 123
Aim 123
Indications 123
Contraindications 123
Instruments 123
Procedure 124
Critical Assessment 124
Flap Operations 124
Aims 124
Indications 124
Contraindications 124
Instruments 124
Various Techniques 125
Procedure for the Modified Widman Technique 126
Postoperative Care 129
Critical Assessment 130
Periodontal Wound Healing 132
General Remarks 132
Presence of Progenitor Cells 132
Reestablishment of a Biocompatible Root Surface 133
Exclusion of Epithelium 133
Wound Stabilization 133
Bone and Bone subsitutes 134
Growth and Differentiation Factors 134
Root Surface Conditioning with Enamel Matrix Proteins 135
Guided Tissue Regeneration 136
Membranes 136
Indications 138
Contraindications 138
Procedure 138
Postoperative Care 139
Critical Assessment 140
Treatment of Furcation-Involved Teeth 141
General Remarks 141
Fundamental Morphological Terms 142
Structures Within Furcations 142
Conservative Furcation Therapy 143
Root Amputation and Hemisection 143
Premolarization 146

Tunnel Preparation 146
Regenerative Procedures 148
Possible Treatment Strategies 151
Mucogingival Surgery 151
General Remarks 151
Widening of the Zone of Keratinized Tissue with a Free Gingival Graft 151
Gingival Recessions 155
Lateral Sliding Flap 155
Coronally Advanced Flap After Vestibular Extension with Free Gingival Graft 156
Coronally Advanced Flap with Connective Tissue Graft 157
Semilunar Coronally Repositioned Flap 158
Envelope Technique 159
Guided Tissue Regeneration 160
Critical Assessment 161
Frenectomy 161
Occlusal Therapy 162
General Remarks 162
Occlusal Splint 163
Occlusal Adjustment 164
Semipermanent Splinting 164
Perioprosthetic Aspects 165

12 Phase III: Supportive Periodontal Therapy 169
Risk Assessment, Risk Communication, Risk Management 169
General Remarks 169
Risk Assessment 169
Local Risk Factors 169
Dentition-Related Risk Factors 170
General Risk Factors 171
Communication 171
Risk Management 172

13 Drug Therapy 174
Antibiotic and Antimycotic Therapy 174
Systemically Administered Antibiotics . 174
Local Administration of Antimicrobial Substances 177
Inhibitors of Tissue Collagenase 179
Antiinflammatory Drugs 179
Nonsteroidal Antiinflammatory Drugs .. 179
Local Glucocorticosteroids 179

References 180

Index 184

Background

1 Anatomy and Physiology

Development

General Remarks

- The term *periodontium* (from the Greek περι, around, and οδονσ, tooth) describes tissues which:
  - anchor the teeth to the bones of the jaws;
  - provide interdental linkage of the teeth within the dental arch;
  - facilitate epithelial lining of the oral cavity in the region of the erupted tooth.
- The developmental, biological, and functional unit of the periodontium consists of four different types of tissues:
  - Gingiva, i. e., the marginal periodontium
  - Root cementum
  - Alveolar bone proper
  - Periodontal ligament
- *Gingiva*, a keratinized soft tissue, surrounds the tooth at the cervical level together with parts of the alveolar process.
- The *desmodontal fiber apparatus* connects the various mineralized forms of *root cementum*, which are in some ways similar to bone tissue, and the *alveolar bone proper*, which is part of the alveolar process.
- The majority of fibers consist of collagen. The fibers insert partly into the inner cortical plate of the alveolar bone and partly into the root cementum.
- The fibers of the periodontal ligament are functionally oriented, and during and after tooth eruption they undergo continuous renewal and remodeling, which is mainly controlled by fibroblasts.
- Those periodontal ligament fibers, which are anchored either in root cementum or in alveolar bone proper, are called *Sharpey's fibers*.
- Oxytalan fiber bundles, which run parallel to the tooth axis, may also be seen. Their function is largely unknown.
- The development of the periodontium is essentially linked to tooth development (Fig. 1.**1**). The number and shape of the teeth are strictly genetically determined. Tooth development is initiated by epithelial thickening of the ectodermal epithelial lining of the

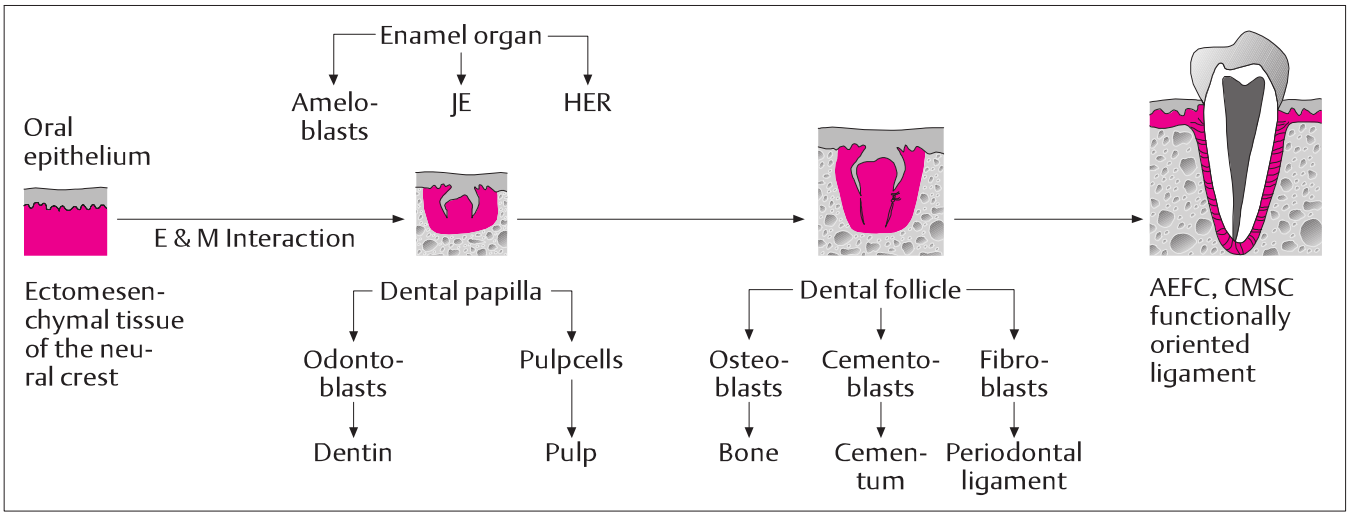


Fig. 1.**1** The development of the periodontium is part of tooth development. Interactions between the epithelium and the ectomesenchymal tissue (*E & M*) of the neural crest beneath lead to the initiation of crown development (enamel, pulp, and dentin). Tissues of the periodontium proper (cementum, alveolar bone proper, periodontal ligament) derive from the dental follicle proper. Proliferation of Hertwig's epithelial root sheath (*HER*) leads to root formation. *JE* junctional epithelium; *AEFC* acellular extrinsic fiber cementum; *CMSC* cellular mixed stratified cementum. (Adapted from McNeal and Somerman 1999)

primitive oral cavity (stomodeum) in the fifth to sixth week of embryogenesis: the odontogenic epithelium, which is called the dental plate or *dental lamina*.

- Originating from the dental lamina, tooth development is controlled by a chain of cell–cell and cell–matrix interactions. Both ectodermal and ectomesenchymal cells reach increasingly higher degrees of differentiation and eventually develop into the highly differentiated ameloblasts and odontoblasts which produce enamel matrix and predentin.

Crown Development

- Between weeks 6 and 8 after ovulation, cells of the dental lamina proliferate in distinct regions (the later positions of deciduous teeth) into the mesenchymal tissue beneath. They induce a condensation of the ectomesenchymal tissue, which derives from the neural crest. This tissue is now called *determinate dental mesenchyme*.
- During morphogenesis of the tooth germ, the *tooth bud* develops between the eighth and twelfth week into the *cap-like enamel organ*. The odontogenic mesenchyme divides into two cell lines:
  - The dental papilla, containing progenitor cells of odontoblasts and the later pulp
  - The dental follicle, which surrounds the tooth germ and develops into the periodontium
- Cells of the enamel organ differentiate into four cytologically and functionally defined strata:
  - Outer enamel epithelium
  - Stratum reticulare
  - Stratum intermedium
  - Inner enamel epithelium
- Finally, the *bell stage* of the tooth germ develops, which already gives an indication of the later shape of the crown.
- The enamel–dentin border is defined when odontoblasts and later ameloblasts differentiate and, in the region of the later cusps and incisal edges of the teeth, start secreting predentin and enamel matrix, respectively. The dental papilla and enamel organ are surrounded by the dental follicle, which demarcates the dental papilla from the surrounding mesenchyme.
- During further odontogenesis the dental lamina of the deciduous molars proliferates distally and becomes committed to the development of the molars of the permanent dentition, which thus belong to the first dentition.
- The tooth germs of the permanent first molars arise between the 13th and 15th week of embryogenesis.
- The germs of the succedaneous teeth arise between the fifth prenatal (central incisors) and tenth postnatal month (2nd premolar), and develop from an apically prolonged secondary dental lamina lingually and palatally to the deciduous germs.

Root Development

- Root formation starts when the dentin and enamel formation reaches the connection between the inner and outer enamel epithelium, the later cementoenamel junction. The final shape of the crown has now been determined.
- Due to further proliferation of the enamel epithelium, the epithelial root sheath (Hertwig's sheath) develops, which is located between the dental papilla and the dental follicle proper:
  - Apically it bends inwards to form the epithelial diaphragm.
  - The double-layered epithelium (outer and inner stratum) is responsible for differentiation of root dentin-forming odontoblasts.
  - It thus, in a manner of speaking, represents a mold for the later tooth root.
- Tooth eruption depends on the prolongation of the dentinal tube, while the diaphragm remains in the same location.
- Coronally, the epithelial root sheath loses contact with the root surface. The sheath disintegrates into a loose mesh of epithelial strands, the epithelial rests of Malassez.

Cementogenesis

- In contrast to the inner enamel epithelium of the enamel organ, the inner enamel epithelium of Hertwig's root sheath does not differentiate into enamel-producing ameloblasts:
  - Cell-cell interactions lead to differentiation of cells of the neighboring ectomesenchy-

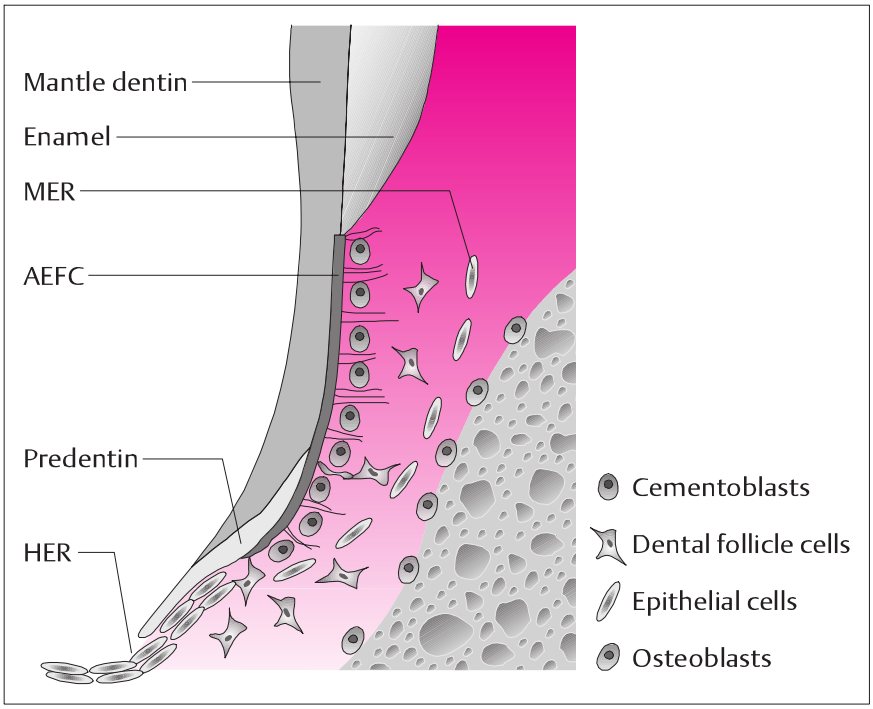


Fig. 1.**2** Initial stages of the formation of acellular extrinsic fiber cementum (*AEFC*). Fibroblasts of the dental follicle come into contact with predentin in the region of the apical edge after disintegration of the epithelial root sheath, attach, and start, after differentiation into cementoblasts, to produce collagen fibrils. An initial fiber fringe with maximum fiber density results. The mineralization front reaches the base of the fibers and proceeds into the initial fiber fringe. (Adapted from McNeal and Somerman 1999)

mal tissue of the dental papilla into odontoblasts, which start forming predentin.
  - Immediately afterwards an enamel-like material is deposited on the surface of the predentin.
  - This material probably induces differentiation of cementoblasts derived from the dental follicle and mediates anchorage of the cement to the dentin surface.
- ➤ Following disintegration of the epithelial root sheath, cells of the dental follicle proper come into contact with the newly formed root surface (cell–matrix interaction). They start the real formation of root cementum (Fig. 1.**2**).
- ➤ Cells and fiber bundles of the periodontal ligament and alveolar bone proper are also derived from cells of the dental follicle proper.
- ➤ These traditional views of cementogenesis have recently been challenged:
  - Presence of mesenchymal cells among disintegrated cells of Hertwig's epithelial root sheet is usually interpreted as a sign for cell migration from the dental follicle proper towards the root surface
  - Alternatively, cells of the root sheet may have completed epithelial-mesenchymal transformation
  - Thus, cementoblasts may originate from Hertwig's epithelial root sheet.
- ➤ In multirooted teeth, shortly after dentin and enamel formation have commenced in the cusp region, two or three epithelial knots are formed in the region of the cervical loop of the enamel epithelium.
  - Tongues of epithelium proliferate across the dental papilla in a central direction.
  - While the size of the enamel organ increases, these epithelial tongues meet and fuse in the region of the future fornix of the furcation.
  - In this way, the future dentin floor of the furcation is created.
- ➤ This means that the formation of the furcation is part of crown development:
  The epithelial root sheath initiates the formation of tooth roots.
  - It determines their shape.
  - In the case of multirooted teeth it divides into two or three branching tubes.
  - The presence of enamel epithelium also explains the frequent formation of *enamel paraplasias* in the furcation area:
    - Enamel projections
    - Enamel tongues
    - Enamel pearls

- Epithelial tongues lie within the connective tissue of the dental papilla and exclude parts of ectomesenchymal tissue from the developing tooth germ. That is why *cementum deposits* (ridges and bulges) are frequently found in the region of fusing epithelial tongues.

Marginal Periodontium

- The epithelial part of the gingiva is of ectodermal origin. Three epithelia can be distinguished:
  - Junctional epithelium
  - Oral sulcular epithelium
  - Oral gingival epithelium
- The *junctional epithelium* derives from the reduced enamel epithelium, which surrounds, in a preeruptive stage, the tooth crown. The reduced enamel epithelium consists of:
  - Ameloblasts, which are reduced in height
  - Cells of the previous stratum intermedium of the enamel epithelium
- The epithelium attaches to enamel in the form of a *primary epithelial attachment* by hemides-mosomes.
- During tooth eruption, reduced enamel epithelium gradually transforms, from coronal to apical, into junctional epithelium (*secondary epithelial attachment*):
  - Reduced cuboid ameloblasts change shape, becoming the elongated cells of junctional epithelium.
  - Cells of stratum intermedium regain their ability to divide and become basal cells of junctional epithelium.
- Posteruptively, junctional epithelium is a self-renewing tissue with specific structures and functions.
- In addition, de novo formation of junctional epithelium is guaranteed:
  - Following a gingivectomy procedure, cells of oral gingival epithelium first migrate to the dentogingival region.
  - Influenced by the underlying connective tissue (i.e., periodontal ligament), these cells develop the characteristics of junctional epithelium:
    - Stratified, two-layered epithelium
    - No keratinization
    - Ability to attach to the tooth surface

Macroscopic and Microscopic Anatomy

Oral Mucosa

- Traditionally, oral mucosa is classified according to function as:
  - Lining mucosa
  - Specialized mucosa
  - Masticatory mucosa
- The nonkeratinized *lining mucosa* comprises:
  - Alveolar mucosa
  - Mucosa of the oral vestibule
  - Mucosa of the cheeks and lips
  - Mucosa of the floor of the mouth and the ventral sides of the tongue
  - Mucosa of the soft palate
- The lining mucosa has a stratified, three-layered, nonkeratinized epithelium:
  - Stratum basale
  - Stratum filamentosum
  - Stratum distendum
- The *specialized mucosa* of the dorsum of the tongue mediates touch, temperature, and taste sensations.
- The keratinized *masticatory mucosa* comprises the gingiva and the mucosa of the hard palate:
  - The gingiva surrounds the teeth and the alveolar bone and extends to the mucogingival border. Palatally, it consists of a small rim, which continues into the mucosa of the hard palate.
  - The structural characteristics of gingival epithelium are essentially the same as those of the mucosa of the hard palate:
    - The gingiva possesses a nonhomogeneous stratum corneum of variable thickness, in which most cells contain a pyknotic nucleus: *parakeratinization*.
    - The mucosa of the hard palate possesses a regularly *orthokeratinized* epithelium with a uniformly thick stratum corneum without pyknotic cell nuclei.
  - The epithelium both of the gingiva and of the mucosa of the hard palate is about 0.3 mm thick, on average.

Fig. 1.**3** Clinical appearance of healthy gingiva

Gingiva

- Clinically, healthy gingiva, i.e., the marginal periodontium, is characterized by certain features of shape, color, and consistency (Fig. 1.**3**):
  - Its narrow band follows the *scalloped contour* of the necks of the teeth and the cementoenamel junction, which is normally covered by gingival tissue. This gives rise to distinct interdental papillae, their vestibular and oral parts being connected by a saddle-like interdental *col*.
  - Gingiva of individuals with a northern European heritage is usually pale pink, coral, or mauve in color. In Mediterranean, African, and Asian populations, melanocytes may give a more or less dark color to the gingiva.
  - Orange-peel-type *stippling* of the surface of the attached gingiva results from indentations lying at the crossing points of rete ridges of the oral gingival epithelium.
  - A frequently observed *gingival groove* separates the free gingiva, which adheres to the enamel surface, from the attached gingiva. The free gingiva ends 1–2 mm on the enamel surface at a narrow angle.
  - A small depression of 0.1–0.5 mm on the tooth surface is called a *gingival sulcus*:
    - The gingival sulcus is bordered by the tooth surface, the oral sulcular epithelium, and the junctional epithelium.
    - *Note:* The depth of the gingival sulcus cannot be determined clinically, e.g., by a periodontal probe.
  - The *mucogingival border* demarcates the gingiva apically.
- Histologically, there are three different epithelia:
  - *Oral gingival epithelium* on the outer surface of the free and attached gingiva
  - *Oral sulcular epithelium* laterally of the gingival sulcus
  - Nonkeratinized *junctional epithelium*, which is located on the inner surface of the free gingiva covering enamel and, in certain situations, root cementum
- Oral sulcular epithelium and oral gingival epithelium are keratinized, stratified, four-layered epithelia (Fig. 1.**4a**) comprised of:
  - Stratum basale
  - Stratum spinosum
  - Stratum granulosum
  - Stratum corneum
- Oral gingival epithelium always contains certain nonepithelial cells:
  - Melanocytes
  - Antigen-presenting Langerhans cells
  - Merkel cells, which operate as sensory mechanoreceptors for touch and pressure reception
  - Small lymphocytes, especially cytotoxic T cells, and, to a minor extent, T helper cells
- Junctional epithelium is not keratinized. It consists of two strata:
  - Stratum basale
  - Stratum suprabasale
- Oral gingival epithelium and oral sulcular epithelium are up to 70–80 % *parakeratinized*, i.e., pyknotic cell nuclei are still found in the stratum corneum. In 20–30 % of cases, the attached gingiva is *orthokeratinized* (Fig. 1.**4a**), i.e., the densely packed horny scales do not contain cell nuclei.
- Junctional epithelium facilitates epithelial lining of the oral cavity during and after tooth eruption.
- The mechanism by which junctional epithelium is attached to different structures of the tooth surface (enamel, cementum, dental cuticle) or implant surface is mediated by:
  - an *internal basal lamina* consisting of glycoproteins and collagen;
  - *hemidesmosomes*.
- Apart from epithelium, gingiva consists of a firm fibrous connective tissue, the lamina propria; there is no submucosa. The supraalveolar fiber apparatus of the lamina propria is composed of primary and secondary fibers

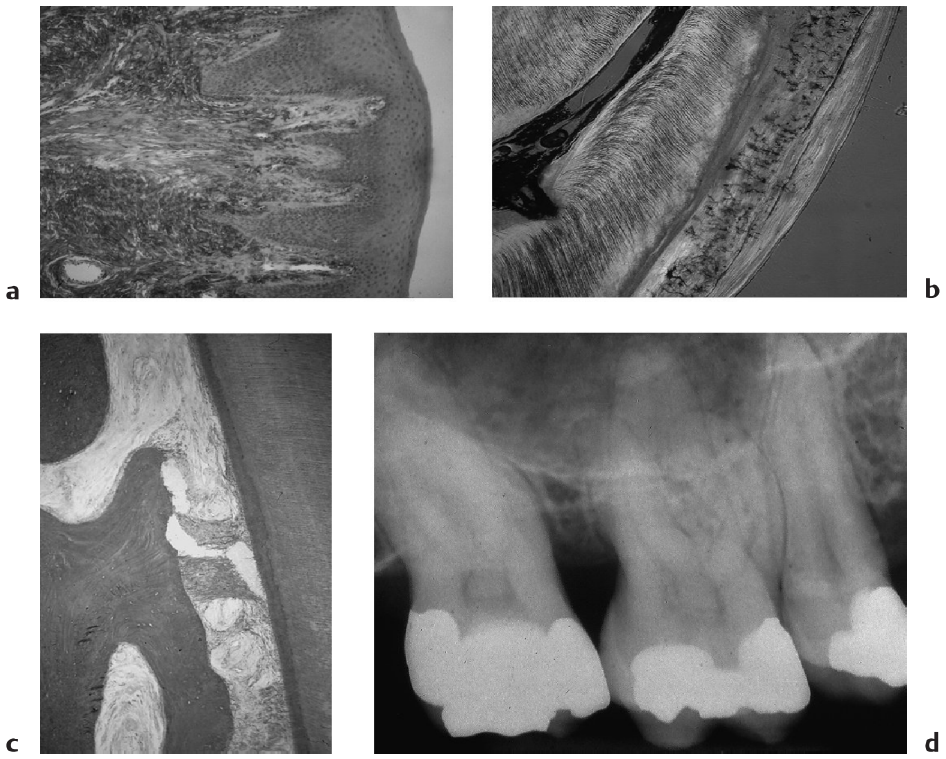


Fig. 1.4 Tissues of the periodontium.
a Lamina propria and oral gingival epithelium with stratum basale, stratum spinosum, stratum granulosum, and stratum corneum. In this case, the epithelium is orthokeratinized.
b Periodontal ligament between alveolar bone proper and acellular extrinsic fiber cementum (AEFC).
c Cellular mixed stratified fiber cementum (CMSC) with layers of cellular intrinsic fiber cementum (CIFC) and AEFC, which covers the surface.
d Alveolar bone proper appears on radiographs as lamina dura (cf. mesial surface of tooth 17)

(Table 1.**1**). The fibers of the secondary fiber apparatus connect the primary fiber bundles.

Root Cementum

➤ Root cementum originates preeruptively during root development and throughout life after completion of root growth. Cementum formation is effected by daughter cells of the ectomesenchymal cells of the dental follicle:
– Cementoblasts
– Cementocytes
– Fibroblasts

Table 1.**1** Composition of the supraalveolar fiber apparatus of the lamina propria

Primary fiber apparatus	Secondary fiber apparatus
Dentogingival fibers	Transgingival fibers
Dentoperiosteal fibers	Intergingival fibers
Alveologingival fibers	Interpapillary fibers
Circular fibers	Periosteogingival fibers
Transseptal fibers	Intercircular fibers
	Semicircular fibers

➤ Various kinds of root cementum have been described depending on histological features and function (Table 1.**2**):
– *Acellular afibrillar cementum* (AAC) is found only on enamel as tongues or islands when enamel has come into contact with connective tissue after the conclusion of crown development. Its function, if any, is unknown.
– The laminar, 20- to 250-µm-thick *acellular extrinsic fiber cementum* (AEFC) is found in the cervical and middle third of the root:
  • AEFC consists of densely packed (30 000/mm^2), about 4-µm-thick, perpendicularly oriented collagen fiber bundles (Sharpey's fibers).
  • Fibers extend into the periodontal ligament and connect the root with the alveolar bone.
  • AEFC is produced first by fibroblasts of the dental follicle proper and is thus of ectomesenchymal origin (Fig. 1.**2**).
  • Later it is produced by fibroblasts of the periodontal ligament.
  • *Note:* AEFC is committed entirely to anchorage of the tooth in its socket.
– *Cellular intrinsic fiber cementum* (CIFC) is a pure product of cementoblasts of the den-

Table 1.2 Forms of human root cementum. (Adapted from Schroeder 1992)

Cementum type	Abbreviation	Organic components	Location	Function
Acellular afibrillar cementum	AAC	Homogeneous matrix, no cells, no fibers	At the cemento-enamel junction, on the enamel	Unknown
Acellular extrinsic fiber cementum	AEFC	Collagen fibrils (Sharpey's fibers), no cells	Cervical up to the middle of the root	Tooth anchoring
Cellular intrinsic fiber cementum	CIFC	Intrinsic collagen fibers, cementocytes	Apical and interradicular root surfaces, resorption lacunae, fracture lines	Adaptation, repair
Acellular intrinsic fiber cementum	AIFC	Intrinsic collagen fibers, no cells	Apical and interradicular root surfaces	Adaptation
Cellular mixed stratified cementum (AEFC + CIFC/AIFC)	CMSC	Intrinsic collagen fibers and collagen fibers as Sharpey's fibers, cementocytes	Apical and interradicular root surfaces	Adaptation, tooth anchoring

tal follicle proper, the later periodontal ligament.
- CIFC contains cementocytes.
- Collagen fibers run circularly or helically around the root, i.e., parallel to the root surface.
- CIFC contains no Sharpey's fibers.
- CIFC is a repair cementum, but it also forms part of cellular mixed stratified cementum (CMSC).

– *Cellular mixed stratified cementum* (CMSC) is a stratified tissue with alternate layers of AEFC and CIFC/AIFC (Fig. 1.**4c**):
- It is inhomogeneously mineralized, partly porous, and of variable thickness (100–600 µm).
- It is mainly found in the apical third of the root and the furcation area of multirooted teeth.
- It is committed to functional adaptation, i.e., dynamic change of the outer shape of the root during movements of the tooth, mesial shift, and occlusal drift.
- If it is covered by AEFC, it anchors the tooth in its socket.
- Infrequently, *acellular intrinsic fiber cementum* (AIFC) is found in CMSC.

Periodontal Ligament

➤ The periodontal ligament is a cell- and fiber-rich, firm connective tissue which anchors the tooth via root cementum and alveolar bone proper in its socket (Fig. 1.**4b**):
- Developmentally it derives from ectomesenchymal cells of the dental follicle proper.
- The periodontal space is narrower in the middle of the root (0.12–0.17 mm) than at the alveolar crest (0.17–0.23 mm) or the tooth apex (0.16–0.24 mm). Higher values are found in adolescents and lower values in older adults.
- Functional strain may lead to a widening of the periodontal space and increasing thickness of collagen fiber bundles.

➤ Desmodontal fiber bundles may be differentiated as follows:
- Supracrestal fibers
- Horizontal fibers
- Oblique fibers
- Interradicular fibers
- Apical fibers

➤ Cellular elements are:
- Fibroblasts
- Cementoblasts and dentoclasts
- Osteoblasts and osteoclasts
- Epithelium (Malassez rests)

- Immune defense cells and neurovascular elements

➤ The periodontal ligament is heavily vascularized. The following may be distinguished:
- The gingival plexus of postcapillary venules
- A desmodontal blood vessel basket, i.e., lateral branches of:
  - the alveolar and infraorbital arteries
  - the lingual and mental arteries

➤ *Lymph vessels* form a dense, basket-like network, which anastomoses with lymph vessels of the gingiva and septa of the alveolar bone.

➤ Both sensory and autonomic nerve fibers are found:
- Somatosensory, afferent fibers reach the periodontal ligament as terminal branches of the dental nerve. Some of them appear apically as lateral branches of the dental nerve whereas others pass through foramina of the cribriform lamina.
- Free nerve endings of sensory fibers are responsible for *pain perception.*
- Ruffini-like endings are *mechanoreceptors* for proprioceptive stimuli, i.e., pressure. Pressure sensitivity is extraordinarily refined.
- Nonmyelinated *sympathetic fibers* are responsible for the local regulation of desmodontal vessels.

Alveolar Bone Proper

➤ Deriving from cells of the dental follicle, the alveolar bone proper is also of ectomesenchymal origin.
- On radiographs it appears as *lamina dura* (Fig. 1.**4 d**).
- Alveolar bone proper contains *Sharpey's fibers*, which are connected to fibers of the periodontal ligament.

➤ Alveolar bone proper may be absent on the vestibular aspects of teeth positioned prominently in the jaw. This condition is termed
- *fenestration* if marginal bone is present and
- *dehiscence* if marginal bone is absent.

➤ Three cell types may be distinguished:
- *Osteoblasts:*
  - Are desmodontal progenitor cells of osteoblasts.
  - Comprise a mixed population composed of preodontoblasts with large nuclei and fibroblast-like cells with small nuclei.
- *Osteocytes:*
  - Arise from osteoblasts, which become entrapped in their own product, i.e., bone.
  - Are located in bony lacunae and are connected by long cell projections.
  - Young osteocytes are smaller than osteoblasts, but have a similar structure.
  - Older osteocytes have a reduced set of organelles.
- *Osteoclasts:*
  - Are large, multinucleated giant cells, which are located in surface pits of the bone (Howship's lacunae).
  - Arise by fusion of hematopoietic, mononuclear precursor cells of bone marrow.
  - An organelle-poor, brush-like cytoplasmic border consisting of microvilli is characteristic.
  - Resorption of bone is facilitated by acidic phosphatases and other hydrolytic enzymes.

Physiology

General Remarks

➤ Soft (gingiva, periodontal ligament) and hard tissues (root cementum, alveolar bone proper) comprising the periodontium are committed to different tasks:
- Anchoring the teeth in their bony sockets
- Holding the teeth together within the jaw as a dental arch
- Adaptation to functional and topographic alterations
- Enabling change in tooth positions
- Repair of the effects of traumatic insult
- Maintenance of the epithelial lining of the oral cavity
- Provision of peripheral defense mechanisms against infection
- Perception of pain and pressure, sensing of touch

Turnover Rates

➤ In *junctional epithelium*, the ratio between the basal cell area and the area of exfoliation results in an extremely high tissue turnover. It is 50–100 times higher than in oral gingival epithelium.

- Tissue turnover of *gingival connective tissue* is higher than that of dermis:
  - Gingival fibroblasts synthesize larger amounts of new collagen than would be necessary for mature collagen replacement.
  - The resulting excess seems to be available for tissue repair.
- *Cementogenesis*:
  - AEFC is formed extremely slowly. In humans, thickening amounts to about 0.005–0.01 µm per day.
  - Initial CIFC is formed considerably faster (0.4–3.1 µm per day). Further layers are built up at a rate of 0.1–0.5 µm per day, which is still faster than AEFC.
  - Growth rates are comparable with those of crown and root dentin, and only slightly slower than the growth of alveolar bone proper.
- The turnover rate of *periodontal ligament* is about twice that of gingiva and four times that of dermis. There is a marked capacity for tissue remodeling. Tissue turnover keeps the structural organization of the tissue constant. During *remodeling*, an adaptation of the three-dimensional organization of the desmodontal fiber apparatus to an altered position or functional strain occurs:
  - Both processes are accompanied by decomposition and synthesis of collagen fibers and are at times indistinguishable.
  - Collagen is removed primarily by *phagocytosing fibroblasts*.
  - Physiological rates of renewal and removal of collagen fibers are balanced.
  - Physiologic forces during chewing stimulate these processes. During aging, turnover decreases.
- Bony remodeling occurs in the *alveolar process* during growth of the jaw, eruption of the teeth, and during tooth replacement. Apposition of bone predominates:
  - Growth starts from periosteum and endosteum.
  - The renewal rate seems to be higher than in other bones.
  - The remodeling of the alveolar bone proper commences with the tooth's functional period, as soon as it comes into occlusal contact with its antagonist.
  - Occlusal forces are transferred to the bone by the periodontal ligament.
  - The direction, frequency, duration, and magnitude of the forces largely determine the extent and rate of remodeling.
- The complicated posteruptive movement of teeth is characterized by an oblique tilting with a vertical and horizontal component:
  - Occlusal drift
  - Mesial migration
  - Eruption following extraction of the antagonist
  - Orthodontic tooth movement

Defense Mechanisms

- The gingiva is protected against mechanical, thermal, and chemical insult by:
  - the firm consistency of the supraalveolar fiber apparatus;
  - the keratinization of the oral gingival epithelium.
- In most circumstances, specific compartments of the peripheral host defense of the gingiva efficiently prohibit bacterial invasion of the dentogingival region:
  - Protection against bacterial infection is provided by both the epithelial and the connective tissue component of the gingiva.
  - Although junctional epithelium does not keratinize, its extreme turnover rate and the presence of resident leukocytes make it relatively resistant to bacterial invasion.
  - The lamina propria of the gingiva provides cellular and humoral components of the immune system.
  - Particularly in young individuals, inflammatory cell infiltrates in the gingiva provide some protection against periodontal destruction.

Possibilities of Repair

- Replantation or transplantation of teeth can be carried out successfully only if cells and fibers of the periodontal ligament on the root surface and the inner surface of the alveolar socket are maintained:
  - Otherwise, ankylosis and/or root resorption will occur.
  - Reparative replacement of desmodontal fibers is carried out by cell populations of the periodontal ligament.

➤ The regenerative potential is, however, essentially limited:
- Reparative, cellular intrinsic fiber cementum may be formed rapidly during wound healing.
- However, this bone-like type of cementum should probably not be regarded as odontogenic tissue.
- The true tissues of the tooth-supporting apparatus (alveolar bone proper, periodontal ligament, acellular extrinsic fiber cementum) are derived from the dental follicle proper, which has its origin in the ectomesenchymal tissue of the neural crest. Differentiation during odontogenesis is dependent on a cascade of genetic signals and growth factors.
- Regeneration of the tooth-supporting apparatus in the proper sense of restoration of the normal tissue architecture should therefore not be expected. The mere reparative deposition of cellular cementum has no functional relevance.

2 Periodontal Microbiology

Ecology of the Mouth

The Oral Cavity as a Biotope

- The oral cavity is a uniquely complex *biotope* in the organism:
  - Hard structures (teeth) interrupt the mucosal lining.
  - Teeth provide *habitats* which allow colonization by specific bacteria:
    - Pits and fissures
    - Smooth surfaces
    - Cervical region of the teeth
    - Root canal system
    - Carious dentin
  - Further habitats include:
    - Periodontal pockets
    - The dorsum of the tongue
    - The tonsils
- Different habitats are colonized by different bacterial communities:
  - On the tooth surfaces: *Streptococcus sanguinis*, *S. mutans*, *Actinomyces viscosus* (*A. naeslundii* genotype 2)
  - On the dorsum of the tongue: *S. salivarius*, *A. naeslundii;*
  - In carious lesions: *Lactobacillus* spp.
  - In the *subgingival region*: spirochetes and motile rods; essentially, obligately anaerobic, gram-negative bacteria
  - Root canal system: obligately anaerobic, gram-negative bacteria
- *Note:* Changes in the ecosystem have a considerable influence on bacterial populations and thus a decisive therapeutic effect. Examples are:
  - Subgingival administration of oxygen, e.g., 3 % H_2O_2;
  - Sealing of fissure systems and establishment of anaerobic conditions without substrate supply

Colonization Mechanisms

- The oral cavity provides very comfortable living conditions for many bacteria:
  - Warm (about 36 °C), humid environment
  - Frequent nutritional supply
  - Firm surfaces to adhere to
- On the other hand, there are defense mechanisms which may interfere with colonization. Bacteria must have certain capabilities if they are to establish themselves in the oral cavity:
  - They must be able to overcome various mechanical obstacles put up by the host:
    - Saliva flow
    - Gingival crevicular fluid flow, which is directed outwards from the gingival sulcus or periodontal pocket
    - Epithelial desquamation
    - Self-cleansing during mastication
    - Personal oral hygiene
  - Both the bacteria and the surface to be colonized are electronegatively charged. Protons and other cations may bridge electrostatic forces.
  - The adhesion of bacteria to the surface is mostly quite specific:
    - Lectin-like adhesins (i.e., proteins that recognize carbohydrate structures of the pellicle [see below]) and hydrophobic adhesins, which react with complementary receptor molecules of the host surface.
    - Adhesins are located in thread-like pili or fimbriae, which can also bridge electrostatic forces and enable contact to the surface of the substrate.
  - Secretory immunoglobulin A (sIgA) of the host and so-called agglutinins may recognize antigenic properties in fimbriae and specifically block them.
  - Colonization by many bacteria further depends on:
    - Redox potential
    - Oxygen tension
    - Antagonisms and synergisms between microorganisms

Biofilm Dental Plaque

➤ A multitude of interactions, e.g., food webs, may exist between different bacteria (Fig. 2.**1**). This might be a main reason for the organization of microorganisms of the dental surface in a highly complex biofilm:
- – Typical bacterial populations colonize firm surfaces in a humid environment, e.g.:
  - • on objects and at the bottom of standing or flowing water;
  - • in the water of sewerage installations.
- – Extracellular structures of a multitude of very various bacteria, such as capsule polysaccharides or glycocalyx, surround the bacterial population as a *matrix*:
  - • protecting bacteria living in a biofilm from external influences;
  - • facilitating survival and growth within the community.
- – Defined microenvironments with differing pH, oxygen tension, and redox potential are characteristic.
- – A primitive *circulation system* supplies substrates and eliminates waste products and metabolites.

➤ Compared to planktonic cultures (bacteria living individually in a fluid culture), bacterial communities in a biofilm (Fig. 2.**2**) exert remarkable properties:
- – Metabolic cooperation
- – A primitive communication system with exchange of genetic information
- – Resistance against phagocytosis and killing by neutrophilic granulocytes, irrespective

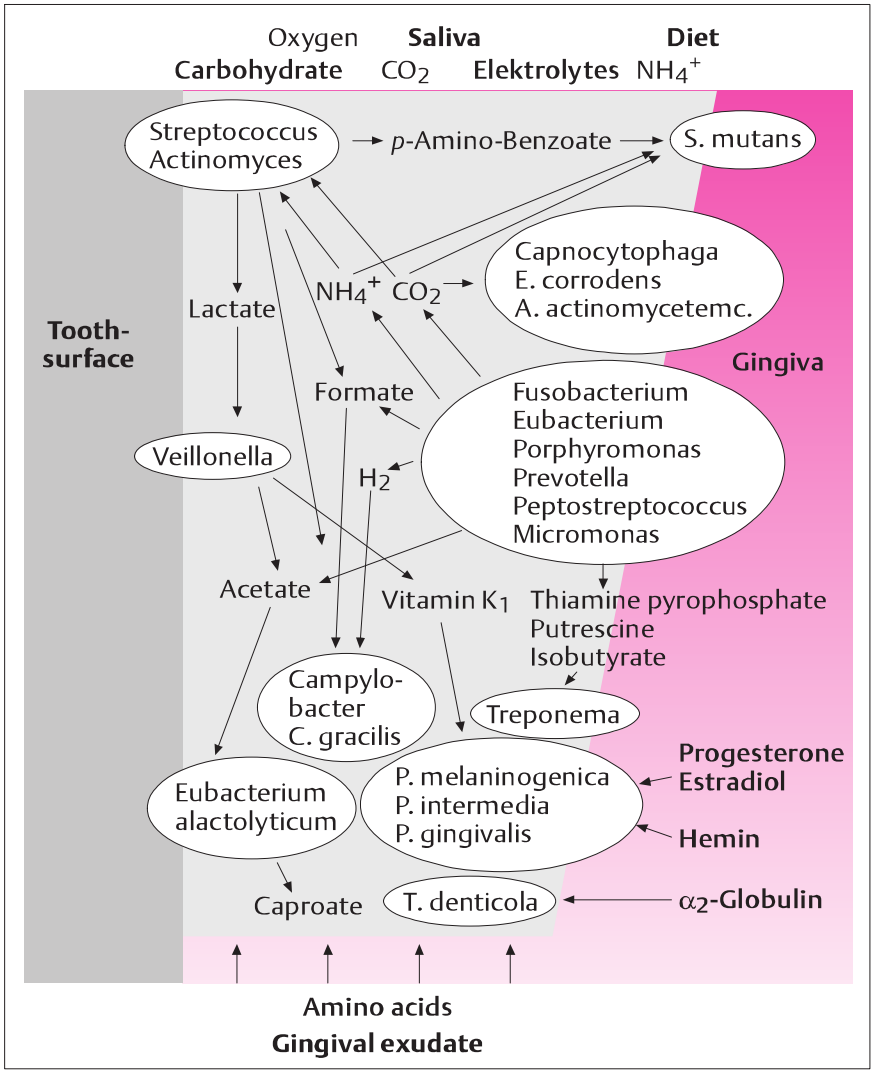


Fig. 2.**1** Bacterial interactions, e. g., food webs, in subgingival biofilms. (Adapted from Carlsson 1989)

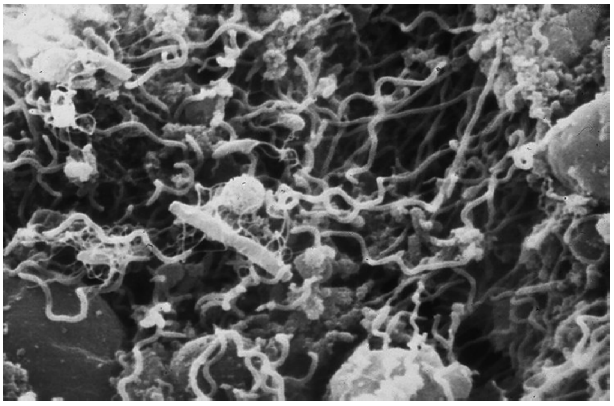


Fig. 2.2 Scanning electron microscopic (SEM) image of the dental biofilm

of presence of specific antibodies and complement
- Resistance against antibiotics due to incorporation in a matrix
- A capacity as a community for drastically increased pathogenicity

Formation of Supragingival Plaque

- With the commencement of plaque formation, i.e., of aggregation of bacteria on the tooth surface, microorganisms of the oral cavity may become pathogenic.
- Within minutes or up to two hours of undisturbed plaque formation, an organic deposition of glycoproteins of saliva is formed on the tooth surface and other hard structures of the oral cavity: the so-called *acquired pellicle*.
- The first bacteria are seen on this pellicle after about four hours:
  - Streptococci, especially *S. mitis*, *S. sanguinis*, and *S. oralis*.
  - Small proportions of gram-positive rods such as *A. viscosus*. These plaque bacteria adhere loosely in the beginning but soon become firmly attached.
  - Most of the bacteria that adhere first to the pellicle are dead.
- The primary adhesion of *S. mutans* on the acquired pellicle is partly due to lectin-like adhesins binding to α-galactoside receptors of salivary glycoproteins.
  - Subsequent production of extracellular glucans promotes further accumulation of these bacteria.
  - The dental plaque is stabilized by *extracellular polysaccharides*, in particular the extremely insoluble 1,3-α-glucan (mutan), which are synthesized by *S. mutans*, *S. sanguinis*, *S. oralis*, and *S. salivarius*.
  - *Aggregations* between streptococci and *Actinomyces* spp. are particularly important for further plaque formation.
  - Salivary bacteria colonize the plaque surface, while bacteria that are only loosely attached are washed away.
  - Irregularities of the tooth surface are preferentially colonized and rapidly leveled.
  - With a generation time of about 0.7–2.4 hours, the main reason for the increase in plaque mass during the first 24 hours is bacterial proliferation.
- If plaque is allowed to accumulate further undisturbed, its composition becomes more and more complex:
  - The proportion of streptococci decreases.
  - The proportions of facultative or obligate anaerobic *Actinomyces* spp. increase.
  - Among gram-negative bacteria, *Veillonella* spp. predominate.
  - Gram-negative anaerobic rods of the genera *Fusobacterium*, *Prevotella*, and *Porphyromonas* appear in small amounts in the supragingival flora.
  - *F. nucleatum* plays a key role in dental biofilm formation because of its multigeneric coaggregation properties.
  - After about one week of undisturbed plaque growth, *spirochetes* and *motile rods* can be observed in the supragingival plaque.
- Thus, plaque formation and maturation go through four phases:
  - Minutes to two hours: pellicle formation (specific adsorption of salivary glycoproteins).
  - First day: gram-positive cocci (*S. mitis*, *S. sanguinis*, *S. oralis*) and rods (*A. odontolyticus*, *A. viscosus*, *A. naeslundii*). Extracellular polysaccharide production (e.g., mutan: 1,3-α-glucan) of *S. mutans*. Leveling of surface irregularities.
  - Second to fourth day: decrease of streptococci, increase of *Actinomyces* spp., gram-negative cocci and rods.
  - After one week: spirochetes and motile rods.

Colonization of the Subgingival Region

- Deepening of the gingival sulcus and edematous swelling of the gingiva as response to supragingival plaque lead to formation of a subgingival space. Later this space may further increase when the junctional epithelium proliferates apically as the result of loss of connective tissue attachment.
- About 500 different bacterial species may live in subgingival sites.
- Most periodontal pathogens of the subgingival region are gram-negative (exceptions, e.g., *Micromonas micros*, *Streptococcus intermedius*, *Eubacterium* spp.) and obligately anaerobic (exceptions, e.g., *Actinobacillus actinomycetemcomitans*, *Eikenella corrodens*, *Capnocytophaga* spp.) (Table 2.1).
- The subgingival region provides very comfortable conditions for many bacteria, but very different from those found in supragingival areas:
  - Protection from oral hygiene measures and saliva flow: this results in selective colonization by bacteria that are unable to adhere to solid surfaces, i.e., in particular, spirochetes and motile rods.
  - Gingival exudate contains nutrients and essential growth factors for numerous periodontal pathogens:
    - Amino and fatty acids as energy source.
    - α_2-globulin, essential for *Treponema denticola*.
    - Hemin, iron, and vitamin K are essential for black-pigmenting, gram-negative an-

Table 2.1 Some gram-positive and gram-negative bacteria of the oral cavity

	Gram-positive		Gram-negative	
	Facultative anaerobes	**Obligate anaerobes**	**Facultative anaerobes**	**Obligate anaerobes**
Cocci	*Streptococcus* • *S. mitis* • *S. oralis* • *S. salivarius* • *S. sanguinis* • *S. gordonii* • *S. vestibularis* • *S. mutans*	*Micromonas* • *M. micros* *Peptostreptococcus anaerobius*	*Neisseria* • *N. sicca* • *N. subflava*	*Veillonella parvula*
Rods/ filaments	*Lactobacillus* • *L. acidophilus* • *L. brevis* • *L. fermentum* *Corynebacterium matruchotii* *Rothia dentocariosa* *Actinomyces* • *A. israelii* • *A. odontolyticus* • *A. naeslundii* • *A. viscosus* • *A. gerencseria*	*Eubacterium* • *E. brachy* • *E. nodatum* *Mogibacterium timidum* *Propionibacterium* *Actinomyces meyeri*	*Haemophilus* • *H. aphrophilus* • *H. parainfluenzae* *Eikenella corrodens* *Actinobacillus actinomycetemcomitans* *Capnocytophaga* *C. ochracea* *C. gingivalis* *C. sputigena* *C. granulosa* *C. haemolytica* *Campylobacter* *C. gracilis* *C. concisus* *C. rectus*	*Porphyromonas* • *P. gingivalis* • *P. endodontalis* • *P. catoniae* *Prevotella* • *P. intermedia* • *P. nigrescens* • *P. pallens* • *P. melaninogenica* • *P. denticola* • *P. loescheii* *Tannerella forsythia* *Fusobacterium* • *F. nucleatum* *Selenomonas* • *S. sputigena* • *S. noxia* *Leptotrichia buccalis*
Spirochetes				*Treponema* • *T. denticola* • *T. vincentii* • *T. socranskii* • *T. pectinovorum*

Table 2.2 Crystalline structures of calcium phosphate in dental calculus

Name	Sum formula	Occurrence
Brushite	$CaHPO_4 \times 2\ H_2O$	Recent calculus
Octacalciumphosphate	$Ca_4H(PO_4)_3 \times 2\ H_2O$	Mostly in outer layers of supragingival calculus
Hydroxyapatite	$Ca_{10}(OH)_2(PO_4)_6$	Mostly in inner layers of supragingival plaque
Whitlockite	$Ca_3(PO_4)_2$	Main component of subgingival calculus; contains small amounts of Mg

aerobes of the genera *Prevotella* and *Porphyromonas*.
  - *Prevotella intermedia, P. nigrescens*, and members of the *P. melaninogenica* group can substitute steroid hormones such as estradiol and progesterone for vitamin K.
  - For this reason, during pregnancy an excessive increase of these microorganisms in gingival pockets is often observed, leading to so-called *pregnancy gingivitis*.
- Favorable conditions for obligately anaerobic bacteria in deep periodontal pockets:
  - Low redox potential (−80 to −100 mV)
  - Low oxygen tension of 13 mmHg, on average.
- Individual diet (frequency and composition of meals) has no apparent influence.
- Specific relationships between periodontal pathogens and beneficial bacteria of the oral cavity (synergisms and antagonisms) may play an important role in the colonization of the subgingival space.

Dental Calculus

➤ Dental calculus is mineralized bacterial plaque:
- *Note:* Dental calculus is not the primary cause of destructive periodontal disease.
- However, dental calculus is always covered by vital plaque; thus, removal of calculus remains central to all periodontal therapy.

➤ The tendency to develop dental calculus and plaque differs among individuals. Mineralization of plaque is brought about by minerals dissolved in the saliva and gingival exudate:
- The partial CO_2 pressure of the saliva rapidly decreases in the oral cavity.
  - This causes the pH to rise.
  - The dissolved minerals are precipitated.
- *Note:* The pH may also rise following production of ammonia from urea.

➤ *Supragingival calculus* is mainly located adjacent to the excretion ducts of the large salivary glands:
- lingually at the mandibular incisors;
- buccally at the maxillary first and second molars.

➤ *Subgingival calculus* covers the tooth/root surface within a gingival/periodontal pocket. Under the antibacterial influence of gingival exudate, a zone at the bottom of the pocket about 0.5 mm wide appears always to be plaque- and calculus-free.

➤ Various crystalline structures of calcium phosphate may be found in calculus (Table 2.2).

Periodontitis: an Infectious Disease

Identification of Periodontal Pathogens

➤ Bacteria cause most of the diseases of the oral cavity. Current concepts of prevention are therefore based on the idea of reducing the bacterial masses in the oral cavity.

➤ However, the main disorders of the oral cavity, i. e., caries and inflammatory periodontal diseases, are not caused by the total bacterial mass or by the plaque in general.

➤ *Note:* Caries and periodontal diseases are usually *endogenous infections*, which depend on certain preconditions:
- The presence of a specific habitat.
- Certain changes in the external conditions promote the increase of particular segments of the complex flora.
- A change in the host's defense system may lead to loss of control over the pathogenic flora in the habitat.

- For a specific pathogen to be identified as the cause of a particular infectious disease, in general Henle-Koch's postulates must be fulfilled, which are as follows:
  - The pathogen occurs without exception in every case of the disease, and under circumstances which can account for the pathological changes and clinical course of the disease.
  - The pathogen occurs in no other disease as a fortuitous and nonpathogenic entity.
  - After being completely isolated from the body and repeatedly grown in pure culture, the pathogen can induce the disease anew.
- Lack of applicability to oral infections prompted Socransky (1979) to modify (and considerably weaken) these postulates as follows:
  - The pathogen is *associated* with diseased sites; it should be absent in healthy sites or in different forms of the disease.
  - *Elimination* of the pathogen should lead to healing of the disease.
  - The following may be taken as significant:
    - The presence of abnormal cellular and/or humoral *immune responses* to the potential pathogen, when responses to other microorganisms are normal
    - Evidence from *animal experiments*
    - The presence of *virulence factors*
- Of paramount importance in epidemiological research (see Chapter 5) are Hill's criteria of causal association:
  - *Strength of association:* what is the relative risk?
  - *Consistency of association:* is there agreement among repeated observations in different places, at different times, using different methodology, by different researchers, under different circumstances?
  - *Specificity of association:* is the outcome unique to the exposure?
  - *Temporality:* does exposure precede the outcome variable?
  - *Biological gradient:* is there evidence of a dose–response relationship?
  - *Plausibility:* does the causal relationship make biological sense?
  - *Coherence:* is the causal association compatible with present knowledge of the disease?
  - *Experimentation:* does controlled manipulation of the exposure variable change the outcome?
  - *Analogy:* does the causal relationship conform to a previously described relationship?
- Based on these criteria the following pathogens were identified:
  - Pathogens strongly associated with periodontal disease:
    - *Actinobacillus actinomycetemcomitans*
    - *Porphyromonas gingivalis*
    - *Tannerella forsythia* (*Bacteroides forsythus*)
  - Potential pathogens moderately associated with periodontal disease:
    - *Treponema denticola*
    - *Prevotella intermedia*
    - *Campylobacter rectus*
    - *Micromonas micros*
    - *Eikenella corrodens*
    - *Fusobacterium nucleatum*
    - *Eubacterium* spp.
    - β-hemolytic streptococci
  - Significant in certain cases:
    - *Staphylococcus* spp.
    - *Pseudomonas* spp.
    - Enterococci, enteric rods
    - *Candida* spp.
- Further developments in molecular biology have led to the formulation of molecular guidelines (presence or absence of a specific nucleic acid sequence) for establishing microbial disease causation.

Types of Infection

- Several kinds of infection can be distinguished (Fig. 2.**3**):
  - *Endogenous infection* with bacteria that belong to the resident flora of the skin, nose, oral cavity, or intestinal and urogenital tract. *Note:* Members of the resident flora at one site may cause life-threatening infections in other organ systems.
    - If oral hygiene is poor, or there are alterations within the habitat or drastic changes in the local or systemic host defense, some of these commensals, i.e., potentially pathogenic bacteria found in every oral cavity, may increase disproportionately.
    - This may lead to periodontitis.

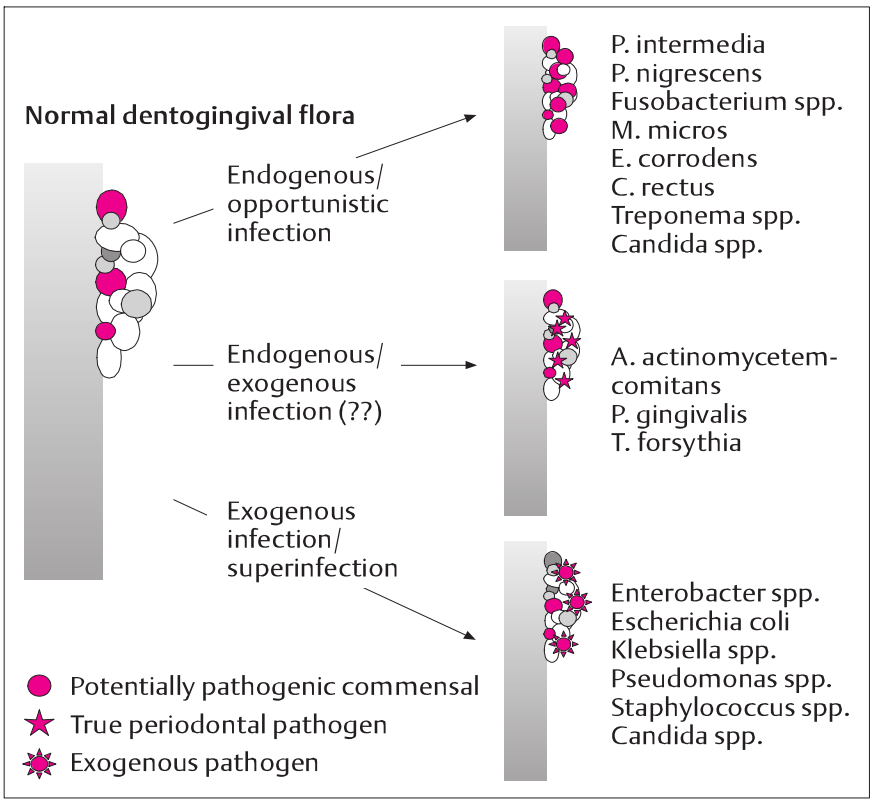


Fig. 2.3 Different types of periodontal infections. Among the normal dentogingival flora, a few, mainly gram-negative, commensals may increase disproportionately in numbers if local conditions in the habitat change, and this may lead to periodontal disease. In an especially susceptible host this is called "opportunistic infection." Virulent variants of some periodontal pathogens such as *Actinobacillus actinomycetemcomitans* and *Porphyromonas gingivalis* are usually not found in the dentogingival biofilm under healthy periodontal conditions. If they enter the oral cavity from outside, they may trigger periodontitis. Normally, the resident microflora of the oral cavity does not include typical exogenous pathogens. However, the former may, if permanently established, trigger periodontitis or accelerate its progression in the form of a superinfection

- *Opportunistic infections* in a systemically or locally compromised host. Opportunistic pathogens are usually not especially virulent.
- *Exogenous infection* with microorganisms that are usually not members of the resident microflora:
  - Among periodontal pathogens, in particular some virulent clones of *A. actinomycetemcomitans* and *P. gingivalis* might be considered exogenous pathogens.
  - Pathogens might be transmitted in the mixed dentition by periodontally diseased parents.
  - A carrier status is frequently found: subjects harboring the pathogen may or may not get the disease.
  - In addition, exogenous infections with enteric bacteria, pseudomonads, staphylococci, etc. are possible. An existing periodontitis may be negatively influenced by presence of these bacteria.

➤ *A. actinomycetemcomitans*, *P. gingivalis*, and *T. forsythia* (*B. forsythus*) are presently regarded as the principal pathogens strongly associated with periodontitis. However:

- Bacteria alone cannot induce destructive periodontal disease.
- Whether periodontal infection actually occurs may depend on at least four factors:
  - A susceptible host
  - The presence of pathogens
  - Absence of beneficial bacteria
  - A conducive environment in the pocket

Table 2.3 Some virulence factors of periodontal pathogens *A. actinomycetemcomitans*, *P. gingivalis*, and *T. forsythia*

	Adhesion/colonization	Evasion/perturbation of host response	Tissue destruction
A. actinomycetemcomitans	• Capsule antigen, pili, vesicle • Antagonistic to *S. sanguinis* and *A. naeslundii* 2	• Leukotoxin • Inhibits chemotaxis • Fc-binding protein • Suppresses lymphocyte functions • Invades epithelial cells in vivo • Degrades Ig	• Endotoxin • Epithelotoxin • Collagenase • Inhibits fibroblast functions • Induces apoptosis • Alkaline, acidic phosphatases
P. gingivalis	• Capsule antigen, pili, vesicle • Synergistic with *T. forsythia*, *T. denticola*	• Inhibits chemotaxis • Degrades complement and Ig • Invades epithelial cells in vitro • Does not trigger expression of E-selectin on inner endothelial surface • Suppresses production and expression of IL-8 and ICAM-1 in junctional epithelium	• Endotoxin • Collagenase • Trypsin-like activity • Fibrinolysin • Gingipain • Phospholipase A • Alkaline, acidic phosphatases
T. forsythia	• Synergistic with *P. gingivalis*, *T. denticola*	• Invades epithelial cells in vivo	• Endotoxin • Trypsin-like activity • Fatty acid production • Induces apoptosis

The Susceptible Host

➤ In many cases of aggressive periodontitis, impaired function of neutrophilic granulocytes, i.e., in particular, chemotaxis and/or phagocytosis, can be observed.

➤ A conspicuously increased susceptibility to periodontal infections is usually associated with systemically or locally excessive, impaired, or inappropriate response to the bacterial challenge:
- – Excessive monocytic secretion of prostaglandin E_2 and interleukin-1β in reaction to outer membrane lipopolysaccharides of some gram-negative bacteria.
- – The systemic component can be an expression of a general mechanism which exposes the individual to an increased risk of chronic inflammatory diseases, i.e., a specific macrophage hyperresponsiveness trait.
- – In this context, some associations between severe forms of periodontal disease and chronic diseases and conditions may be considered:
  - • Coronary heart disease, coronary infarction
  - • Nonischemic stroke
  - • Low birth weight
- – Systemic diseases such as inadequately controlled type I and type II *diabetes mellitus* or infection with the human immunodeficiency virus (HIV) increase the risk of periodontitis.
- – Hormonal effects of psychological stress:
  - • Increased susceptibility to certain inflammatory periodontal diseases, such as necrotizing ulcerative gingivitis/periodontitis.
  - • Both stress and strategies for coping with stress are also associated with chronic periodontitis.
- – Genetic factors (see below).
- – Smoking (see below).

Presence of Pathogens

➤ Periodontal infection with bacteria such as *A. actinomycetemcomitans*, *P. gingivalis*, or

T. forsythia (*B. forsythus*) frequently leads to the production of specific antibodies in the host:
- In response to antigens of gram-negative bacteria of the subgingival biofilm especially, specific antibodies of the IgG2 subclass are produced.
- In localized aggressive periodontitis, titers of specific antibodies against *A. actinomycetemcomitans* may be as high as those against *Treponema pallidum* in tertiary syphilis: 0.1–1 µg/ml. These antibodies usually protect the organism from developing generalized aggressive periodontitis.

➤ To become pathogenic, bacteria must show certain *virulence factors* (Table 2.**3**):
- Adhesion to tissues of the host
- Growth and multiplication within the habitat
- Evasion and/or perturbation of host-derived defense mechanisms
- Active destruction of colonized tissue

➤ The expression of virulence factors may vary considerably within a given species, being:
- partly genetically determined;
- dependent on growth conditions.

➤ An especially toxic variant of *A. actinomycetemcomitans*, which produces considerable amounts of leukotoxin, has so far been identified only in individuals with a North-West African affiliation (Africans, African Americans, Afro-Indians):
- Oral infection with this highly leukotoxic clone (Fig. 2.**4**) considerably raises the risk of aggressive periodontitis.
- Toxic clones of *A. actinomycetemcomitans* have not been identified in individuals of Northern European or Asian heritage.

➤ The destructive effects of the various virulence factors of different pathogens add up within the environment of the periodontal pocket:
- Leukotoxin and cytotoxin of *A. actinomycetemcomitans*
- Bacterial collagenase
- Endotoxin (lipopolysaccharide)
- Factors which suppress growth and proliferation of fibroblasts or activate osteoclasts
- Trypsin-like peptidases of *P. gingivalis*, *T. forsythia* (*B. forsythus*), and *T. denticola*
- Fibrinolysin and other proteolytic enzymes
- Acidic and alkaline phosphatases
- Toxic substances such as hydrogen sulfide (H_2S), ammonia (NH_3), or fatty acids

Absence of Beneficial Microorganisms

➤ Bacteria and their products are necessary and beneficial components of a healthy periodontium.

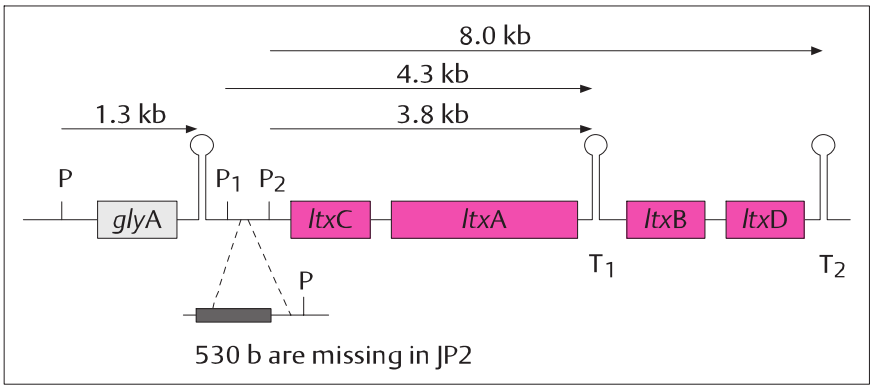


Fig. 2.**4** Gene map of the leukotoxin gene of *A. actinomycetemcomitans*. The membrane-bound leukotoxin belongs, together with the hemolysin of *Escherichia coli* and the leukocidin of *Pasteurella haemolytica*, to the family of RTX toxins, which cause pore formation of the cell membrane of neutrophilic granulocytes and monocytes. The operon consists of four genes: ltxC is responsible for activation of the primary product of ltxA, which decodes the toxin itself. ltxB and ltxD decode proteins for intracellular transport. As compared to minimally leukotoxic strains, the highly toxic strain JP2 is characterized by a 530 base pair deletion in the promoter region of the leukotoxin gene. These strains produce 10–20 times higher amounts of leukotoxin than minimally leukotoxic strains. An intraoral infection considerably increases the risk of development of aggressive periodontitis. (Adapted from Lally et al. 1996)

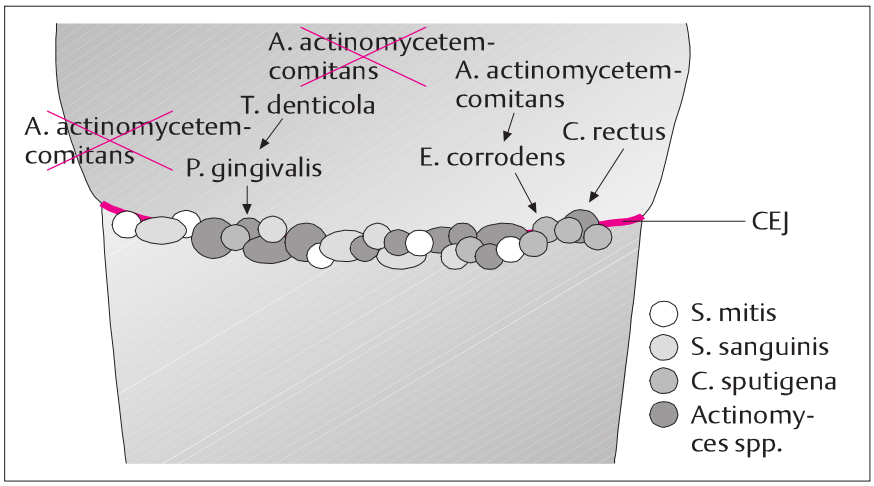


Fig. 2.**5** Influence of some beneficial bacteria (e. g., *Streptococcus mitis*, *S. sanguinis* II, *Capnocytophaga sputigena*, or *Actinomyces* spp.) at the cementoenamel junction (*CEJ*) on the establishment of periodontal pathogens (e. g., *P. gingivalis*, *A. actinomycetemcomitans*, *Eikenella corrodens*, *Campylobacter rectus*, *Treponema denticola*). For instance, *A. actinomycetemcomitans* cannot colonize if *S. sanguinis* is already present (antagonism). If, on the other hand, *C. sputigena* has formed colonies, *A. actinomycetemcomitans*, *C. rectus*, and *E. corrodens* may establish (synergisms). *T. denticola* has no chance in the presence of *A. actinomycetemcomitans*. (Adapted from Socransky et al. 1988)

➤ In the course of evolution, gram-positive bacteria have adapted more closely to the conditions in the human oral cavity, and can sometimes keep certain less robust periodontal pathogens in check (Fig. 2.**5**):
- Antagonistic microorganisms may have an effect on the ability of pathogenic bacteria to successfully establish themselves in the periodontal pocket.
- Identification of these (beneficial) bacteria is based on:
  - frequent demonstration of their presence in healthy periodontium or following successful treatment of periodontitis;
  - their presence during inactive phases of periodontitis.
- The presence of these bacteria appears to interfere in a concentration-dependent manner with the establishment of typical periodontal pathogens. Especially important are:
  - *Actinomyces* spp.
  - *Streptococcus mitis*
  - *S. sanguinis* II
  - *Capnocytophaga* spp.
  - *Veillonella parvula*
- Mutual antagonisms play an important role:
  - *S. sanguinis* inhibits growth of *A. actinomycetemcomitans* by producing H_2O_2.
  - A bacteriocin of *A. actinomycetemcomitans* interferes with growth of a number of streptococci.

Conducive Environment in the Periodontal Pocket

➤ The ecological conditions in the region of the gingival sulcus and periodontal pocket may vary considerably. They partly determine the complex composition of the microflora, which consists mainly of obligately anaerobic, gram-negative, rather fastidious bacteria (Fig. 2.**6**).

➤ Many pathogenic microorganisms regulate expression of available virulence factors according to certain environmental signals.
- Specific responses to the present ecological condition allow colonization and invasion of the host organism. Of importance are:
  - pH
  - Redox potential
  - Oxygen tension
  - Osmotic pressure

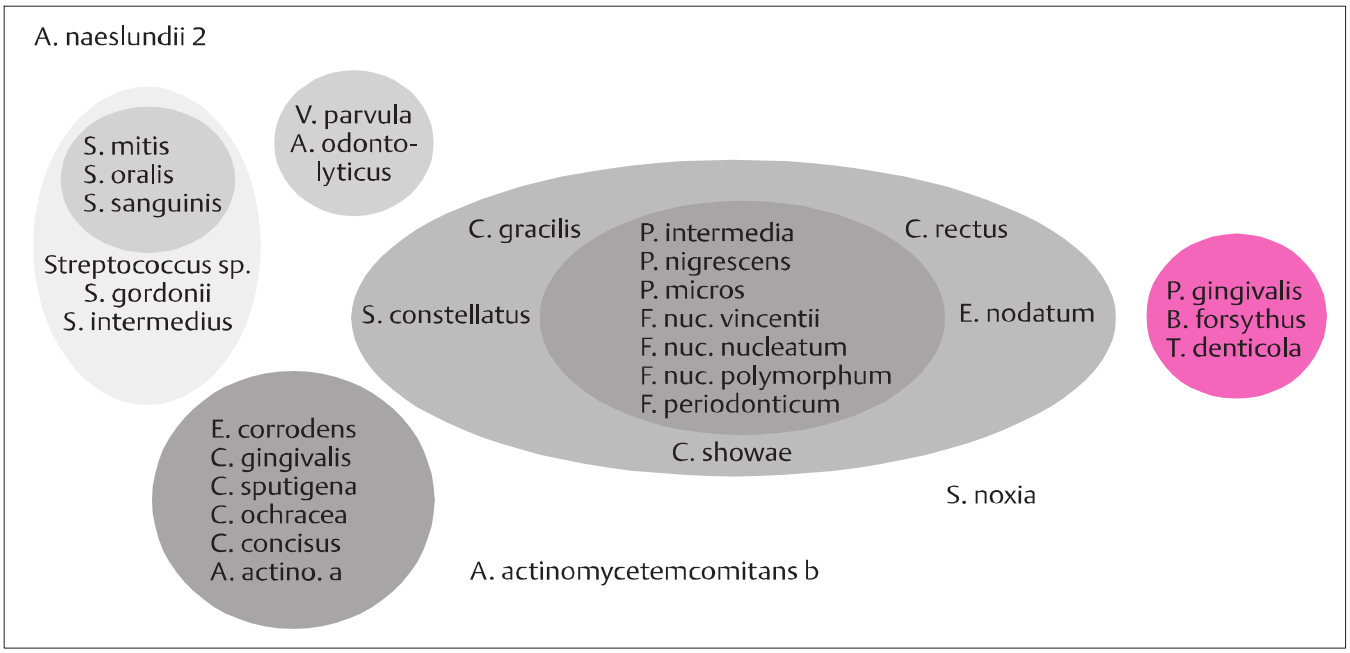


Fig. 2.**6** Bacteria of the oral cavity are organized in different complexes. The figure also indicates the temporal sequence of colonization of the dental surface, from *left* (primary colonizers *A. naeslundii* 2, which occurs most frequently, and streptococci) to *right*. The large complex in the *middle* contains potential periodontal pathogens (*Prevotella* spp., *Fusobacterium* spp., *Campylobacter* spp.) which are usually present at low concentrations in all oral cavities; only in response to changes in their ecosystem will they multiply excessively and cause pathological conditions. The *red* complex is comprised of periodontal pathogens (*P. gingivalis* and *Tannerella forsythia* [*Bacteroides forsythus*]) which are strongly associated with destructive periodontal disease. Serotype b of *A. actinomycetemcomitans* may be an outsider within the complex flora. Serotype a is associated with the complex containing *E. corrodens*, *Capnocytophaga* spp., and *Campylobacter concisus*. (Adapted from Socransky et al. 1998)

- Temperature
- Iron, magnesium, and calcium concentration
- Antibody titers

– Absence of oxygen, for instance, increases leukotoxin production by especially virulent strains of *A. actinomycetemcomitans* more than three-fold.
– The availability of iron, on the other hand, does not seem to play an important role in the regulation of leukotoxin expression by *A. actinomycetemcomitans*.
– However, the amount of available iron does determine the expression of certain outer membrane proteins of *P. gingivalis*.

➤ The strong dependence of the virulence of periodontal pathogens on conditions in their respective habitat may partly explain the long latency between first colonization and outbreak of the disease, i.e., the periodontal infection.

Transmission

➤ The main causes of caries and periodontal diseases—mutans streptococci, *A. actinomycetemcomitans*, and *P. gingivalis*—can be transmitted between individuals.
– The window of infectivity for mutans streptococci, for instance, is at the age of around 24 months.
– The earlier infection takes place, the more caries are to be expected at the age of, say, 4 years.

➤ This has important consequences for primary prevention of caries. If carious conditions in the maternal oral cavity can be resolved before tooth eruption in the child, such that the likelihood of infection of the neonatal child decreases because of low levels of mutans streptococci in the oral cavity of the mother, caries development in the deciduous and permanent dentitions will be delayed, re-

duced, or even prevented (so-called primary primary prevention).

- It is possible to change the flora in the mother in a targeted way by:
  - establishment of effective oral hygiene;
  - change of diet;
  - temporary eradication of mutans streptococci by chlorhexidine-containing varnish;
  - treatment of open carious lesions;
  - consistent follow-up supportive care.
- This program is also effective in prevention of pregnancy gingivitis.
- Transmission of periodontal pathogens from the periodontally diseased mother or father to their child is also possible.
  - *A. actinomycetemcomitans* may be acquired in the mixed dentition; stable colonization by *P. gingivalis* occurs only during adolescence.
  - These and other potential periodontal pathogens are found in numerous ecosystems outside the periodontal pocket:
    - Saliva
    - Dorsum of the tongue
    - Tonsillar region
  - Transmission of *A. actinomycetemcomitans*, *P. gingivalis*, and other periodontal pathogens is mainly through, e.g., common use of toothbrushes or cutlery, or exchange of saliva:
    - vertically, from periodontally diseased parent to child;
    - probably also horizontally, between adult partners.
- *Note:* At least the virulent variants of *A. actinomycetemcomitans* and *P. gingivalis* may be regarded as exogenous pathogens which considerably increase the risk of disease development if transmitted early in life.

3 Pathogenesis of Plaque-Induced Periodontal Diseases

Pathogenesis of Plaque-Induced Gingivitis

General Remarks

- Complete lack of any inflammatory cells in gingival tissues—i.e., *histologically healthy gingiva*—is achieved only by prolonged and total absence of any microbial plaque; this is only possible under experimental conditions.
- *Clinically normal gingiva* has several characteristic features. There is almost always an infiltrate composed of polymorphonuclear granulocytes and lymphocytes.
  - In contrast to other tissues, endothelium of the gingival vascular plexus expresses *adhesion molecules* such as E-selectin.
  - There is therefore a continuous influx of polymorphonuclear granulocytes leaving the plexus and migrating through the connective tissue and junctional epithelium into the sulcus.
  - In even a periodontally healthy human individual, about 500 000 leukocytes reach the oral cavity every minute.
- Acute inflammatory reactions are associated with gingival redness, edematous swelling, and exudation. These lesions can be painful,

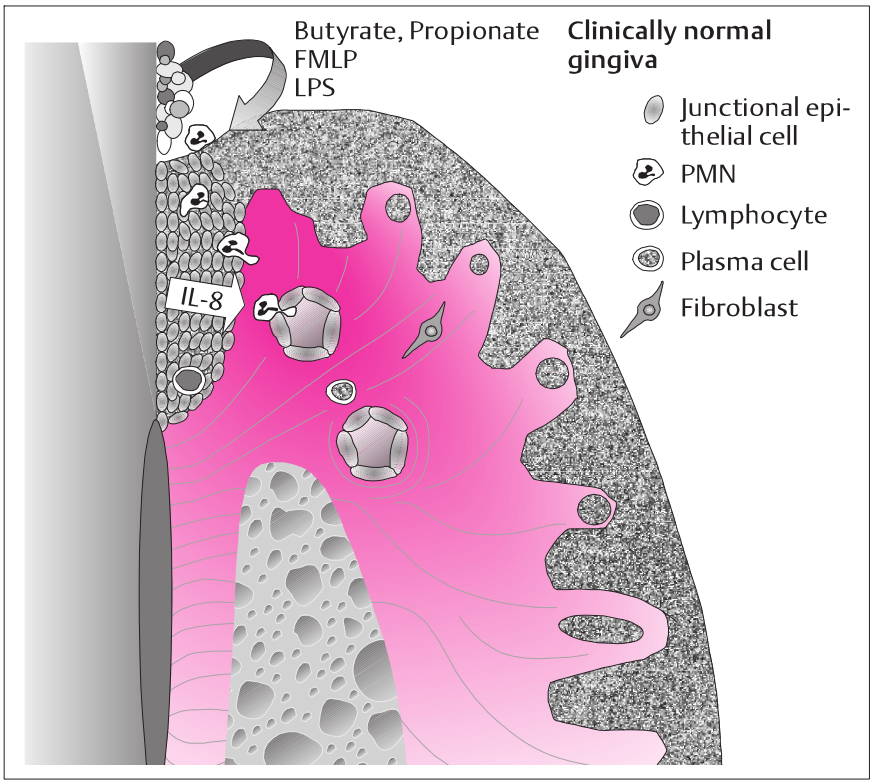


Fig. 3.1 Components of clinically normal gingiva. Histologically, an initial lesion is found. Bacteria of supragingival plaque produce metabolites such as butyrate, propionate, chemotactic peptides such as *N*-formyl-methionyl-leucyl-phenylalanine (FMLP), and lipopolysaccharide (LPS). These trigger junctional epithelial cells to releasing the chemokine interleukin 8 (IL-8). A chemotactic gradient into junctional epithelium and sulcus leads polymorphonuclear granulocytes to migrate from the vascular plexus. Scattered lymphocytes and plasma cells are seen. (Adapted from Page and Schroeder 1990)

especially if ulceration occurs. Causes of *acute gingivitis* are:
- Nonspecific trauma:
  - Thermal burning
  - Chemical burn
  - Mechanical insult
- Bacterial plaque

➤ Histologically, different stages of plaque-induced gingivitis may be distinguished:
- Initial gingivitis
- Early gingivitis
- Established gingivitis

➤ Traditionally, inflammatory processes are divided into three phases:
- An *acute phase*, which is characterized by invasion of neutrophilic granulocytes after tissue injury. Endogenous mediators influence the acute reaction:
  - Bradykinin and prostaglandins lead to *vasodilatation*.
  - Histamine and leukotrienes increase *vascular permeability*.
  - Complement and leukotrienes serve as *chemotactic factors*.
- The *immunologic phase* commences with the arrival of antigen-presenting cells.
- *Chronic lesions* are characterized by an increase of antibody-secreting plasma cells.

➤ *Note:* The classical phases of acute and chronic inflammation cannot be easily differentiated in inflammatory periodontal diseases:
- Acute signs of inflammation (e.g., exudation) are found even in clinically normal gingiva.
- Afterwards, acute and chronic (immunologic) inflammatory components coexist.

Initial Gingivitis

➤ Within 2–4 days after commencement of plaque accumulation, specific alterations in the junctional epithelium and the vascular plexus beneath may be observed. Clinically, the gingiva appears to be absolutely healthy: normal gingiva (see Fig. 1.**3**). Pathohistologically, acute signs characteristic of an *initial lesion* are visible (Fig. 3.**1**).
- Increase of vascular permeability close beneath the junctional epithelium with loss of perivascular collagen.
- Exudation of plasma proteins.
- Large numbers of polymorphonuclear granulocytes migrate through the junctional epithelium into the sulcus.
- In consequence, intercellular spaces widen in the most coronal part of the junctional epithelium.
- The granulocyte migration leads to an increase in the gingival exudate of levels of leukotriene B_4—a product of degranulating neutrophils.

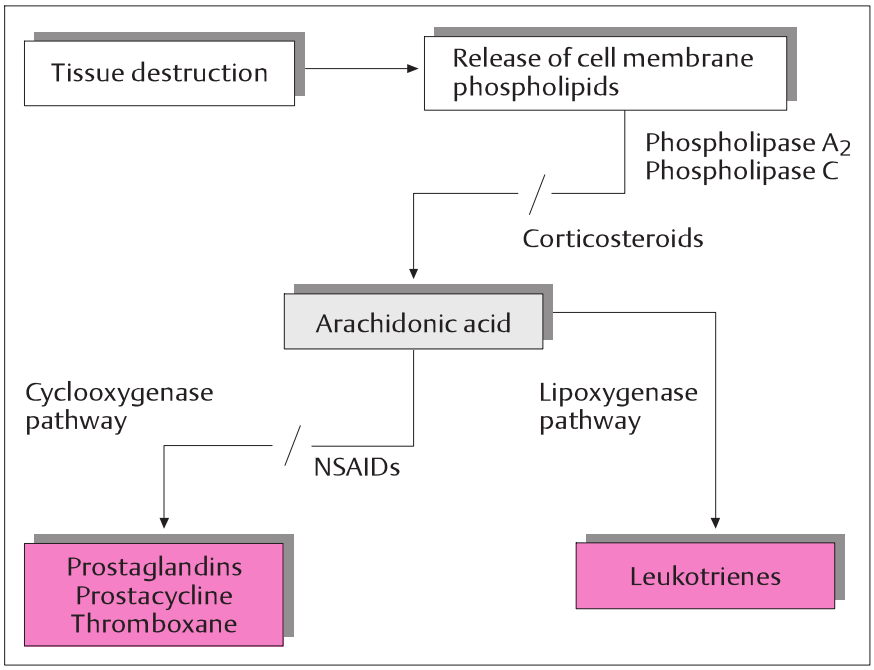


Fig. 3.**2** Metabolites of arachidonic acid play an important role in inflammatory reactions of several chronic diseases. Arachidonic acid is usually esterized with phospholipids of the cell membrane. When tissue is destroyed, phospholipases are activated and arachidonic acid is released. Glucocorticosteroids prevent activation of phospholipases. Prostaglandins, prostacycline, and thromboxane originate after activation of cyclooxygenases COX1 and COX2 (inhibition by nonsteroidal anti-inflammatory drugs, NSAID), whereas lipoxygenase leads to formation of leukotrienes. Many metabolites of arachidonic acid exert important biological activities (Table 3.**1**).

Table 3.**1** Biological functions of arachidonic metabolites

Metabolite	Function
Prostaglandins	• Vasodilatation • Pain • Platelet aggregation • Modulation of T lymphocytes, inhibition of cytokine production • Stimulation of osteoclasts
Leukotrienes	• Increase of vascular permeability • Enable adhesion of polymorphonuclear granulocyte (PMN) to endothelial cells • Stimulate chemotaxis of PMN and eosinophilic granulocytes • Induce lysosomal degranulation of PMN • Enhance chemokinesis of T lymphocytes

- Endogenous mediators are responsible for initiating inflammation:
  - Vasoactive amines (histamine, serotonin)
  - Plasma proteases (kinin, complement, and plasmin systems)
  - Metabolites of arachidonic acid: prostaglandins, leukotrienes (Fig. 3.**2**) with numerous biologically important functions (Table 3.**1**)
- The 20 proteins of the *complement system* form (like the clotting system, fibrinolysis, and kinin formation) a triggered plasma enzyme system:
  - These systems respond rapidly, in an amplifying reaction cascade, to a triggering factor.
  - The product of each reaction is an enzymatic catalyzer of the next.
- Complement activation takes place via:
  - the *classical* pathway, which requires antigen antibody complexes;
  - the *alternative* pathway, which may be induced by microbial lipopolysaccharides (Fig. 3.**3**).
- The main component is C3, a protein with a molecular weight of 195 kDa and a plasma concentration of 1.2 mg/ml:
  - Under normal circumstances and in the presence of Mg^{2+}, the larger fragment C3b, which is formed spontaneously at a very slow rate, may complex with complement component factor B. Following cleavage by plasma enzyme factor D, $C\overline{3bBb}$ is generated, which exerts enzymatic activity as a C3 convertase.
  - The C3 convertase $C\overline{3bBb}$ is stabilized on the bacterial surface by the serum protein properdin and activated by microbial polysaccharides.

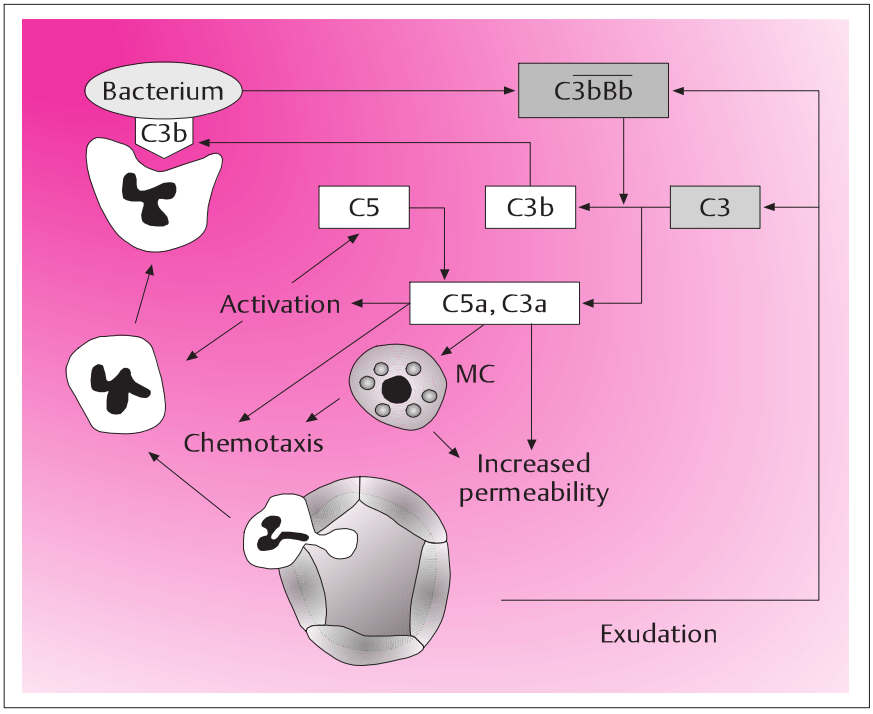


Fig. 3.**3** Acute inflammatory reaction by activation of the alternative pathway of the complement cascade. First, C3 convertase $C\overline{3bBb}$ is stabilized on the bacterial surface and cleaves large amounts of C3 into fragments C3a and C3b. C3b binds to the bacterium; C3a activates C5. The cleavage product C5a is a chemotactic factor for polymorphonuclear neutrophils (PMN), which leave the vessel and migrate directly to the bacterium. Mast cells release vasoactive mediators, which result in a further influx of PMN and complement into the tissue

- C3 convertase cleaves large amounts of C3 into fragments C3a and C3b.
- C3b binds to the bacterium.
- C3a activates C5, which is cleaved into C5a and C5b.

➤ Complement components C3a and C5a exhibit several defense mechanisms:
- C5a is a strong chemotactic factor for polymorphonuclear granulocytes
- C3a and C5a are anaphylatoxins in that they are capable of triggering:
  - mediator release from mast cells;
  - release of vasoactive mediators such as histamine, leukotriene B_4 (increase of vascular permeability), and prostaglandins (vasodilatation).

➤ Further activation of the complement cascade leads to pore formation in the cell membrane with subsequent bacterial lysis:
- C5b binds loosely to C3b.
- C6 and C7 bind successively to C5b.
- After formation of C8a and C9 the membrane attack complex is formed.

➤ For leukocytes to leave the vascular plexus, both leukocytes and endothelial cells must express adhesion molecules on their respective surfaces:
- Bacterial products such as lipopolysaccharides (LPS) of gram-negative bacteria or proteins may react *directly*, in the form of split-off vesicles, with endothelial cells of capillaries and venules of the gingival connective tissue.
- *Indirect* activation by macrophages is also possible. After contact with LPS or bacterial proteins, these cells secrete proinflammatory cytokines such as interleukin-1 (IL-1) and tumor necrosis factor-α (TNF-α).
- In both cases, the adhesion molecule E-selectin is expressed on the inner surface of the endothelial cell, which causes granulocytes to roll on the endothelium.
- After contact between the leukocyte adhesion receptor CD11/18 and the endothelial integrin ICAM-1 (intercellular adhesion molecule 1), the leukocyte can leave the vessel by amoeboid diapedesis (Fig. 3.**4**).

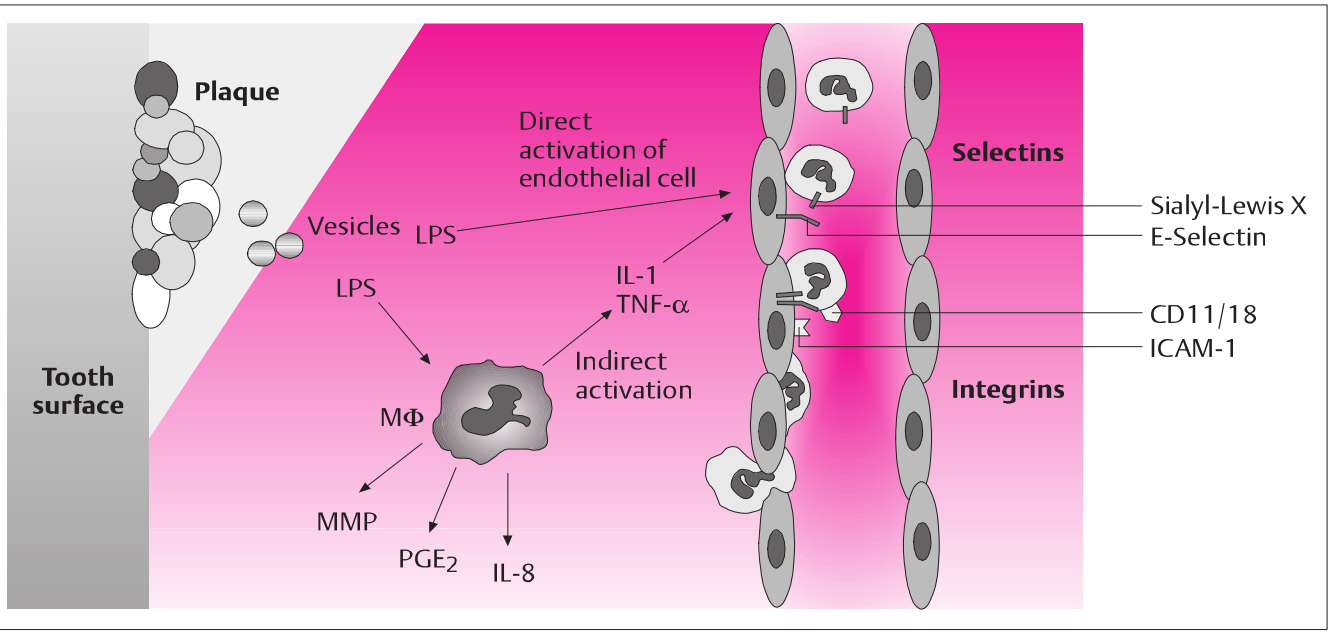


Fig. 3.**4** Activation of endothelial cells may be triggered directly by split-off vesicles of LPS of gram-negative bacteria residing on the tooth surface, or indirectly by proinflammatory cytokines interleukin-1 (IL-1) and tumor necrosis factor α (TNF-α), which are released by macrophages (MΦ) together with other cytokines, matrix metalloproteinases (MMP), prostglandin E_2 (PGE_2), and chemokine IL-8. E-selectin is expressed on the inner surface of the vessel. It binds loosely by Lewis blood group antigen to granulocytes. The velocity of the latter in the blood stream is dramatically slowed. As soon as granulocytes start to roll on the endothelium, stronger adhesion comes into effect by the leukocyte adhesion receptor CD11/18 and the intercellular adhesion molecule ICAM-1. After this, the granulocyte migrates out of the vessel. (Adapted from Darveau et al. 1997)

- The migration of granulocytes follows the gradient of chemotactic factors:
  - Bacterial peptides such as *N*-formyl-methionyl-leucyl-phenylalanine (FMLP)
  - Chemotactic cytokines (chemokines) such as IL-8, which is released in large amounts by cells of the junctional epithelium
  - Leukotriene B_4, which is released by other granulocytes
  - Complement component C5a
- For migration, the granulocytes need a sufficient number of functioning surface receptors.
- Apart from its barrier function, junctional epithelium has *sensory* and *signaling* functions:
  - It expresses the integrin ICAM-1. This allows neutrophilic granulocytes and certain lymphocytes to adhere by leukocyte adhesion receptor CD11/18, signaling the presence of bacteria to the connective tissue below.
  - Both junctional and pocket epithelium possess neural elements.
- When polymorphonuclear granulocytes reach the sulcus, complement and antibodies (opsonins) support phagocytosis of bacteria and their products.
- Leukocytes bind to opsonized bacteria by Fc receptors:
  - FcγRI binds with high affinity to immunoglobulins of the IgG1, IgG3, and IgG4 subclasses.
  - FcγRII and FcγRIII bind with low affinity to antigen–antibody complexes or aggregations of IgG1 and IgG3.
  - *Note:* FcγRII is the only Fc receptor binding to IgG2, the IgG subclass formed mainly against polysaccharide capsule antigens of gram-negative bacteria.
- After phagocytosis, phagosomes fuse with cytoplasmic granules (Table 3.**2**) and become phagolysosomes. Bacteria are killed intracellularly:
  - *Oxygen-dependent killing:*
    - Superoxide anion: $NADPH + 2\ O_2 \xrightarrow{\text{oxidase}} NADP + H^+ + 2\ O_2^-$
    - Hydroxyl radical: $O_2^- + 2\ H_2O \longrightarrow 2\ H_2O_2 \longrightarrow \cdot OH$
    - Myeloperoxidase-mediated: hypochloric acid, chloramines
  - *Oxygen-independent killing:*
    - Myeloperoxidase
    - Defensins: specific antibiotic peptides
    - Cathepsins
    - Lactoferrin
    - Lysozyme
- Extracellular delivery of the granular content for defense against invading bacteria usually results in considerable tissue destruction:
  - Endopeptidases (proteases) are inhibited by α_2-macroglobulin and α_1-proteinase inhibitor.
  - On the other hand, these inhibitors are easily cleaved by potent proteases of perio-

Table 3.**2** Cytoplasmic granules of polymorphonuclear granulocytes

Enzymes	Primary (azurophilic) granula	Secondary (specific) granula
Microbicidal enzymes	Myeloperoxidase Lysozyme	Lysozyme
Neutral proteases	Elastase Cathepsin G Proteinase 3	Collagenase
Acidic proteases	*N*-acetyl-β-glucosamidase Cathepsins B, D β-Glucuronidase β-Glycerophosphatase β-Mannosidase	
Other	Defensins Cationic proteins Bactericidal/permeability increasing factor	Lactoferrin Cobalophilin CR3 Cytochrome

dontal microorganisms; e.g., gingipain by *P. gingivalis*.

- The concentration of several plasma proteins, so-called acute phase reactants, increases under the influence of proinflammatory mediators such as IL-1:
  - Acute phase reactants that increase enormously in concentration:
    - C-reactive protein (CRP): binds complement, opsonizes.
    - Mannose-binding protein: binds complement, opsonizes.
    - Serum amyloid P: initiates amyloid deposition.
  - Acute phase proteins that increase moderately in concentration (protease inhibitors):
    - α_2-Macroglobulin
    - α_1-Proteinase inhibitor

Early Gingivitis

- Within 2 or 3 weeks of undisturbed plaque accumulation some cardinal symptoms of inflammation, such as redness and swelling of the gingiva, become clinically visible. These alterations are due to:
  - increasing vascularity;
  - enhanced permeability of vessels with extravasation of plasma proteins.
- This leads to a strong increase in gingival crevicular fluid flow, which is clinically detectable.
- The *early gingival lesion* represents the mounting of a competent immune reaction against plaque antigens:
  - It is the typical lesion found in healthy children and adolescents.
  - It has similarities to *lymphoid tissue*.
- This immune reaction is launched by cells residing in the junctional epithelium:
  - Special mucosal T lymphocytes
  - Antigen-presenting *Langerhans cells*
  - *Dendritic cells*
- The infiltrate residing in the connective tissue consists mainly of T lymphocytes. It occupies about 10–15% of the volume of the free gingiva (Fig. 3.**5**).
- B lymphocytes are observed in only small numbers. They differentiate into antibodies secreting plasma cells.
- Systemic humoral immune reaction:
  - Antigenic material from the bacteria is taken up by epithelial Langerhans cells and macrophages residing in the connective tissue of the gingiva, and is transported to regional lymph nodes.
  - Stimulation of lymphocytes, which mount a specific immune response:
    - Plasma cells residing in lymph nodes produce specific antibodies.
    - Antibodies enter the circulation and arrive at the gingiva.
    - These specific antibodies can then be detected in gingival crevicular fluid.
- Whether a specific immune response is also mounted locally is not as clear:
  - In this case, B and T cells *highly specific* for periodontal tissues would have to proliferate in regional lymph nodes and enter the circulation.
  - These lymphocytes would then settle in the periodontium and start to exercise their respective humoral and cellular functions.
  - Plasma cells under the control of Th2 cells produce antibodies. Th1 cells (see below) regulate cell-mediated immune responses.
- *Macrophages* always comprise only a small proportion of the cell population:
  - They become effector cells after LPS exposition and secrete:
    - proinflammatory cytokines;
    - prostaglandin E_2 (PGE_2);
    - chemokines;
    - matrix metalloproteinases such as collagenase.
  - In this way, continuous recruitment of lymphocytes and macrophages is facilitated.

Established Gingivitis

- In adults, further accumulation of bacterial plaque results, after an indeterminate period of time, in development of *established gingival lesions*.
- A gingival pocket forms as a result of:
  - *Intraepithelial tear* with subsequent degeneration of junctional epithelial cells
  - Loss of the biological connection between junctional epithelium and enamel surface
  - Development of a *pocket epithelium:*
    - Proliferation of epithelial ridges, which extend into the infiltrated connective tissue

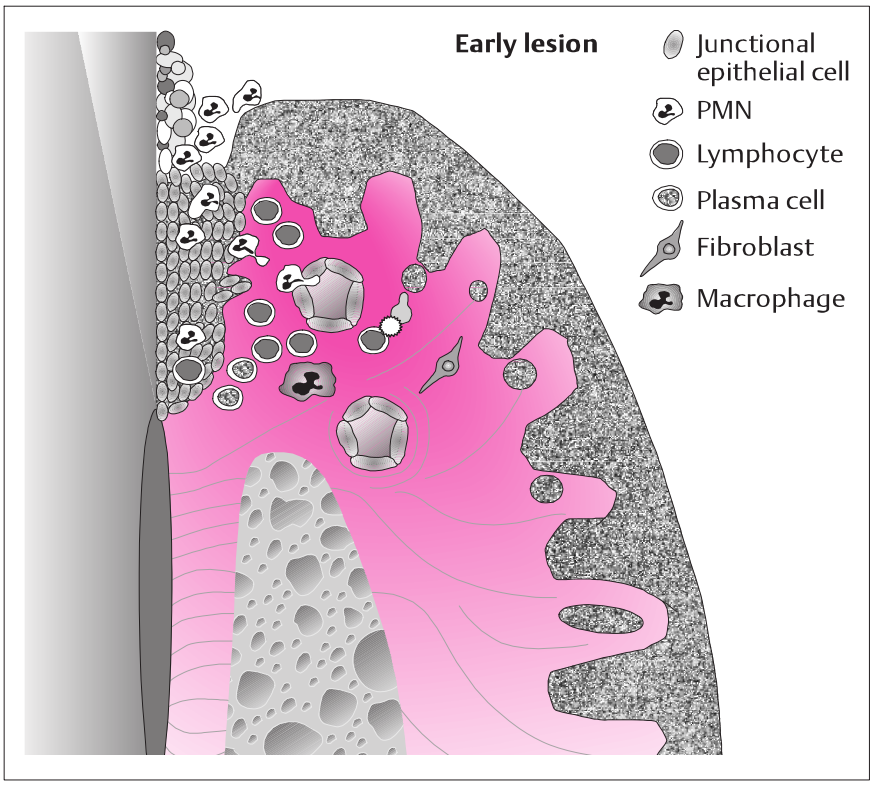


Fig. 3.**5** Characteristics of the early lesion. Further increase in vascular permeability leads to a strong influx of plasma proteins including acute phase reactants, complement, and plasmin. Large numbers of PMN pass the connective tissue and junctional epithelium, which expresses the chemokine IL-8. This is accompanied by structural alterations of the sulcus bottom, and lateral proliferation of basal cells. Activated macrophages produce proinflammatory cytokines IL-1, IL-6, IL-10, TNF-α, chemokines as IL-8 and the monocyte chemoattractant protein 1 (MCP-1), as well as PGE_2 and tissue collagenase, thus ensuring recruitment of additional T lymphocytes and monocytes from the venule plexus. T lymphocytes interact with fibroblasts, which exhibit cytopathic alterations. The infiltrate of the early lesion occupies about 10–15 % of the volume of the free gingiva. (Adapted from Page and Schroeder 1990)

- *Microulceration* between epithelial ridges
- Some remains of junctional epithelium at the bottom of the gingival pocket

➤ Apical proliferation of bacteria results in the establishment of a *subgingival microflora*. Bacterial metabolites can now directly affect the connective tissue beneath.

➤ Specific, very various populations of inflammatory cells migrate into the connective tissue and pocket:
- Neutrophilic granulocytes form a dense wall against microorganisms.
- Macrophages, lymphocytes, and plasma cells form the main cell populations in the connective tissue. There is selective immigration of:
  - antigen-specific memory cells;
  - activated lymphocytes;
  - mucosal, γδ-receptor-positive T cells;
  - CD1a-positive antigen-presenting cells.
- Selective expression of adhesion molecules plays a major role in the regulation of specific migration.

➤ Chemotactic cytokines with a low molecular weight and potent, cell-type-specific characteristics (chemokines) are especially important:
- IL-8 specifically reacts with neutrophilic granulocytes and a small population of lymphocytes.
- MCP-1 (monocyte chemoattractant protein 1) is responsible for migration of macrophages.

➤ Antibodies produced by plasma cells are not only directed towards antigens found in plaque. Rather, plaque bacteria seem to in-

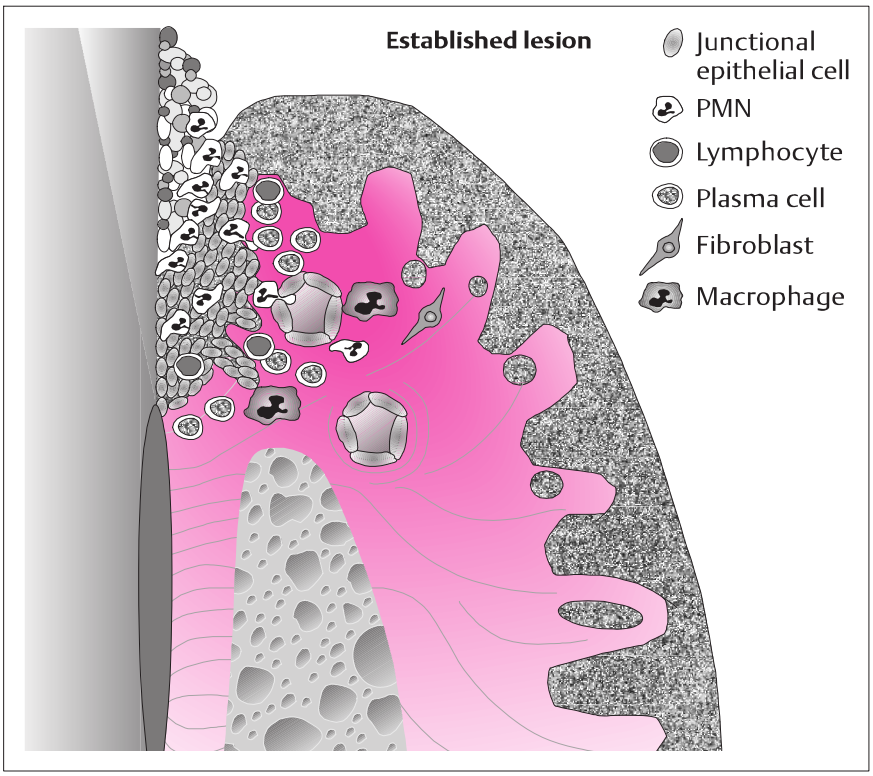


Fig. 3.**6** Characteristics of the established lesion. Distinct lateral proliferation of junctional epithelium. An intraepithelial tear leads to gingival pocket formation and, subsequently, subgingival proliferation of bacteria. Acute components of inflammation persist. Specific populations of mononuclear cells dominate in the infiltrate. In addition to T lymphocytes, B lymphocytes and plasma cells occur, which produce γ-globulins, mainly nonspecific, polyclonal antibodies. (Adapted from Page and Schroeder 1990)

duce an unspecific, polyclonal B cell response.

- Established gingivitis is an extremely frequent condition. Its lesions may be found virtually in every adult's mouth.
- It is not exactly known how long it takes for typical established lesions to develop if plaque is allowed to accumulate, but speculations range between a few weeks and several months.
- Pathohistologically, established lesions are characterized as follows (Fig. 3.**6**):
  - Persistent acute components of inflammation
  - Specific populations of inflammatory cells in the infiltrate
  - Immunoglobulins in extravascular connective tissue and junctional epithelium
  - Increasing proportion of plasma cells
  - Further loss of collagen
  - Lateral proliferation of junctional epithelium and gingival pocket formation
- Established gingivitis may remain quite stable for prolonged periods. Usually, there is a delicate balance between the bacterial challenge and the immune response of the host. After an indeterminate period of time, an *advanced lesion* (periodontitis) may develop.
- Thus, there are two forms of established gingivitis:
  - In most cases it is an independent, self-contained, stable lesion.
  - Infrequently it is a precursor of periodontitis.
- These two forms cannot be distinguished clinically. Pathohistologically, active lesions may be characterized by a higher density of plasma cells (more than 50%).

Pathogenesis of Periodontitis

Advanced Lesion

- Continuous challenge by subgingivally colonizing bacteria may result, after an indeterminate period, in breakdown of specific and nonspecific defense mechanisms of the host:
  - Initial attachment loss seems to be associated with an increase of *T. forsythia*, *C. rectus*, and *Selenomonas noxia* in subgingival plaque.

Table 3.3 Origin and function of some cytokines

Cytokine	Origin	Function during inflammation
Interleukin-1 (IL-1)	Macrophages, fibroblasts	• Proliferation of activated B and T cells • Induction of PGE_2 and cytokine production of macrophages • Expression of endothelial adhesion molecules • Induction of IL-6, INF-β1, and granulocyte-macrophage colony-stimulating factor (GM-CSF) production • Induction of fever, acute phase reactants, osteoclast activity
Tumor necrosis factor-α (TNF-α)	Macrophages, T cells	• Induction of acute phase reactants • Activation of phagocytes • Induction of IFN-γ, TNF-α, IL-1, GM-CSF, IL-6
Interferon-γ (INF-γ)	T cells, natural killer cells (NK)	• Induction of Th1 (T helper) cells • Inhibition of IL-4 activity • Enhances production of IL-12 • Stimulates activity of macrophages, cytotoxic T cells, and NK
IL-2	T cells	• Stimulates growth of activated T and B cells, NK
IL-4	T cells, mast cells, basophilic granulocytes	• Induces differentiation of Th2 cells • Inhibits activities of IL-2 and INF-γ • Inhibits production of IL-12 • Induces proliferation and differentiation of B cells • Induces proliferation of T cells • Downregulation of monocytic production of IL-1, TNF-α, and IL-6
IL-5	T cells, mast cells	• Proliferation of activated B cells • Production of IgM and IgA
IL-6	Th cells, macrophages, mast cells, fibroblasts	• Growth and differentiation of B and T cells • Induction of acute phase reactants
IL-10	T and B cells, monocytes and macrophages	• Enhancement of Th2 reactions and concomitant suppression of Th1 reactions • Suppression of proliferation and cytokine production of activated T cells • Suppression of monocyte production of IL-1, IL-6, and IL-8 • Enhancement of IL-1ra production • Enhancement of proliferation and differentiation of B cells
IL-12	B cells, macrophages, dendritic cells, keratinocytes, neutrophilic granulocytes	• Key role in induction of Th1 reactions • Stimulates growth and cytotoxic activity of NK and T cells
IL-13	T cells	• Downregulation of IL-12 production • Mounting of Th2 reaction • Stimulation of B cells • Inhibition of cytokine production of macrophages

  - Active phases with clinically apparent loss of periodontal attachment occur infrequently and are probably of short duration.

➤ Further progression of the lesion (and, concomitantly, of the front line of plaque) beyond the cementoenamel junction leads to the stage of periodontitis, in which all structures of the periodontium are involved:

  - Definite establishment of a *subgingival plaque microbiota.*
  - Proliferation of the thin pocket epithelium, with its bizarre epithelial ridges, is accompanied by apical proliferation of the remains of junctional epithelium on the cementum surface, which is covered by degenerate fibers of the supraalveolar fiber apparatus.
  - Root cementum is pathologically altered and may be penetrated by bacteria.

➤ While inflammation becomes more severe, the concentration of cytokines decreases:

- Neutrophils migration gradually wanes.
- Granulocytes may be activated even in the connective tissue, leading, if there is a local deficiency of α_2-macroglobulin and α_1-proteinase inhibitor, to excessive periodontal destruction.

➤ T lymphocytes may be differentiated according to their surface antigens:
- *T helper cells* (Th: CD4+) bind to antigens of the MHC-II complex and recognize epitopes presented by corresponding cells. Regulation of cell-mediated and humoral immune response by different cytokines (Table 3.**3**):
  - Th1 cells produce interferon-γ (INF-γ) and IL-2. Cell-mediated reactions and delayed hypersensitivity; stimulation of neutrophilic granulocytes and activation of macrophages; production of proinflammatory cytokines including IL-1 and TNF-α.
  - Th2 cells produce IL-4, IL-5, IL-6, and IL-10. Effect on antibody-mediated and allergic reactions; differentiation of B cells to plasma cells; stimulation of mast cells and eosinophilic granulocytes.
  - Th0 cells produce INF-γ, IL-2, IL-4, and IL-5.
- *Cytotoxic/suppressor T cells* (Tc, Ts: CD8+) bind class I antigens of the MHC complex:
  - Cytotoxic Tc cells produce IL-10 and INF-γ.
  - Ts suppressor cells produce IL-4.

➤ In periodontitis, the ratio between CD4+ and CD8+ cells in the inflammatory infiltrate of the gingiva is increased. Among helper cells, Th2 cells seem to predominate:
- Their cytokines amplify the local humoral immune response.
- In particular, B lymphocytes are stimulated by IL-4 to secrete IL-1.
- In contrast, IL-1 production by macrophages is suppressed.

➤ Under the influence of LPS and proinflammatory cytokines and mediators such as IL-1, TNF-α, or PGE_2, junctional epithelial cells, fibroblasts, and vascular endothelia exert genetically programmed destructive activities:
- In health, genes for the production of collagen and inhibitors of matrix metalloproteinases (MMP) are activated, while genes for production of tissue collagenase are inactivated
- Under pathologic conditions this is reversed.
- If stimulated by LPS, fibroblasts secrete IL-1β by themselves.

➤ Monocytes/macrophages might play a central role. The activity of macrophages is mainly genetically determined. It can be suppressed by INF-γ.
- LPS of gram-negative and lipoteichoic acid from gram-positive bacteria bind, via lipopolysaccharide binding protein (LBP), to the CD14 receptor of macrophages
- If transferred to Toll-like receptors (TLR) that are capable of transmitting a signal into the cell via their IL-1 receptor-like domain:
  - A signal is transferred through the membrane
  - Nuclear transcription factor (NF)-κB, a key regulator of immune and inflammatory responses, is activated
  - Production and secretion of prostaglandins, cytokines and MMP is initiated
- Alternatively, LBP or soluble CD14 (sCD14) may transfer LPS and lipoteichoic acid to nonactivating serum proteins, e.g. serum lipoprotein, for eventual removal of the offending bacterial components from the system without local activation of the innate host response.
- TNF-α and IL-1β bind to surface receptors of fibroblasts, inducing the production of MMP and PGE_2:
  - MMP leads to destruction of the extracellular matrix of gingiva and periodontal ligament
  - PGE_2 activates osteoclasts and induces bone resorption
  - IL-1β and TNF-α are also involved in bone destruction.

➤ Innate immunity provides not only a first line of antimicrobial host defense, but also has a profound impact on the establishment of adaptive immune responses
- Innate host responses, characterized by pattern recognition receptor binding to microbial components, form a highly coordinated and dynamic process
  - Toll-like receptors (TLR) link innate and acquired immunity
  - They may represent the most ancient host defense system found, for example, in mammals, insects, and plants.

- TLR recognize and, more significantly, discriminate between, different classes of pathogens.
  - TLR expression repertoire varies on different cell types:
    - Endothelial cells express more TLR4 (responds to LPS from gram-negative bacteria) than TLR2 (ligand for, e.g., lipoteichoic acid from gram-positive bacteria)
    - In contrast, due to lack of TLR4 ligands, intestinal epithelia do normally not respond to LPS.
  - Bacterial components and host signaling molecules may influence TLR expression.
- Some periodontal pathogens possess virulence factors that interfere directly with defense mechanisms of the host:
  - *A. actinomycetemcomitans* produces:
    - leukotoxin, which kills neutrophilic granulocytes, monocytes, and T lymphocytes;
    - a low-molecular-weight protein which suppresses chemotaxis of neutrophilic granulocytes;
    - immunosuppressive factors which suppress production of IgG and IgM;
    - Fc-binding protein which competes with specific receptors of neutrophilic granulocytes and prohibits phagocytosis.
  - *P. gingivalis:*
    - Does not trigger expression of E-selectin on the endothelial inner surface.
    - Its LPS only weakly activates IL-1 and TNF-α, cytokines which indirectly induce expression of E-selectin.
    - Suppresses production, respectively expression, of IL-8 and ICAM-1.
    - Produces enzymes that cleave most serum proteins including immunoglobulins.
  - Immunodominant molecules (gingipain, fimbrillin, heat-shock proteins, and other surface antigens) produced by *P. gingivalis* and other periodontal pathogens may induce excessive immune reactions.
  - Saliva plays an important role at the mucosal level in the maintenance of oral health.
    - High antibody titers of IgA1 and large amounts of secretory IgA2 against oral pathogens such as *A. actinomycetemcomitans*, *P. gingivalis*, or *S. mutans* can prevent the development of inflammatory conditions in the gingiva or dental caries.
    - However, periodontal pathogens may cleave IgA1 and IgG.
  - Serum titers of specific antibodies, especially IgG, against *A. actinomycetemcomitans* and/or *P. gingivalis* are considerably increased in some periodontally diseased patients with subgingival infection. If antibody production does not occur, periodontitis is frequently more generalized.
  - Pathohistologically, the advanced periodontal lesion (periodontitis), has the following characteristics (Fig. 3.**7**):
    - Persistence of all characteristics of the established lesion
    - Affection of alveolar bone and periodontal ligament
    - Formation of a periodontal pocket
    - Further loss of collagen in the area of the periodontal pocket, fibrosis in more distant areas
    - Marked predominance of plasma cells, especially in active lesions
    - Transformation of bone marrow into fibrous connective tissue

Formal Pathogenesis—Progression

- Inflammatory periodontal diseases may occur in an isolated fashion or at various sites and teeth in the oral cavity.
- The disease is characterized by progressive loss of parts of the tooth-supporting apparatus which usually proceeds apically and is limited laterally:
  - Loss of connective tissue attachment of supraalveolar fibers
  - Loss of supportive alveolar bone
- Although periodontitis manifests itself locally, there is undoubtedly a systemic component as regards both its pathogenesis and its influence on the body's health:
  - Whether destructive periodontitis develops or not mainly depends on the ability of the organism, in conjunction with neutrophilic granulocytes, antibodies, and complement, to prevent exposure of the connective tissue to dentogingival plaque bacteria, their metabolites and, especially, LPS:
    - In this case, only a mild, self-limited form of the disease will occur.

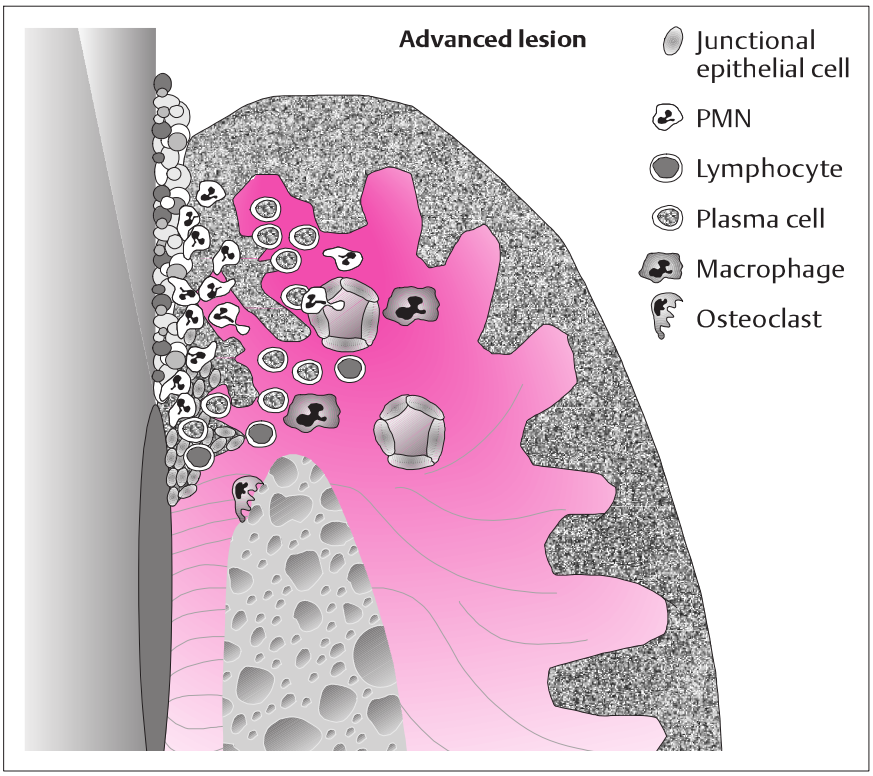


Fig. 3.**7** Characteristics of the advanced lesion. Pocket epithelium with bizarre epithelial ridges. Loss of connective tissue attachment to the tooth. The ratio of T helper cells to cytotoxic T cells is increased. Th2 cells produce cytokines which amplify the humoral immune response especially. Therefore, plasma cells predominate in the inflammatory infiltrate. Bone loss is induced by osteoclasts, which are activated by high concentrations of IL-1β and PGE_2. (Adapted from Page and Schroeder 1990)

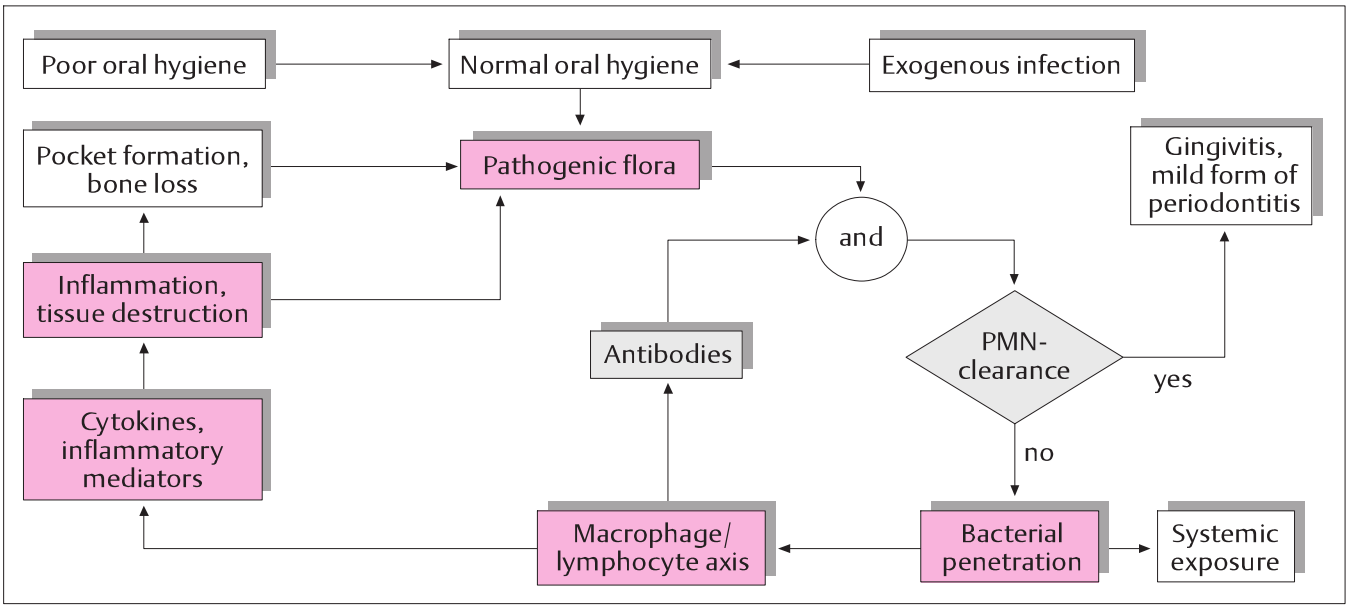


Fig. 3.**8** Pathogenesis pathway of periodontitis. Pathogenic flora develops due to either neglect of oral hygiene or exogenous infection. Usually, the first line of defense, i. e., the granulocyte/complement axis, prevents penetration of bacteria or their metabolites into connective tissue. Gingivitis develops. If penetration does occur, the macrophage/lymphocyte axis is activated. Specific antibodies are produced which have a protective role during later episodes of the disease. Proinflammatory cytokines and mediators such as IL-1 or PGE_2 lead to increased inflammation with tissue destruction and, consequently, pocket formation and bone loss. Pockets provide excellent conditions for the growth of most periodontal pathogens. (Adapted from Offenbacher 1996)

- Subsequent production of antibodies will in most cases localize the disease.

– Destructive periodontal disease may be the result of an activation of the macrophage/lymphocyte axis (Fig. 3.**8**).

➤ Some individuals respond to dentogingival plaque with an immediate activation of tissue macrophages and lymphocytes:
– Macrophage hyperresponsiveness trait:
  - is probably genetically determined;
  - may be influenced by smoking, stress, and some dietary factors;
  - results in high concentrations of IL-1 and PGE_2 in gingival connective tissue and crevicular fluid.
– During phases of undisturbed plaque accumulation the gingivitis stage will rapidly further develop into destructive periodontitis in these individuals.

➤ An important characteristic of chronic diseases is the succession of progression and remission:
– On certain tooth surfaces periodontitis probably advances in a continuous manner.
– Progression rates vary; exacerbations with notable loss of attachment within a short time are relatively rare.
– After destructive phases, there may be prolonged periods of remission.

Characteristics of a Multifactorial Disease

➤ As in most chronic diseases, there are initiating factors, factors that impede development, and factors that influence the clinical expression.
– Bacteria produce a number of substances that have a deleterious effect on the host.
– *Note:* The larger part of periodontal tissue destruction is due to the inflammatory and immune reactions of the host.
– Specific periodontal pathogens explain no more than 20 % of the clinical variability of inflammatory periodontal disease. Smoking, for instance, has a greater impact.
– Inflammatory and immunological reactions and the metabolism of connective tissue and bone are actually genetically controlled, but influenced by acquired and behavioral risk factors (Fig. 3.**9**).

➤ Acquired and behavioral risk factors include:
– Type I and type II diabetes mellitus (relative risk between 2 and 3)
– Smoking (relative risk between 2.5 and 6, even higher in adolescents)
– HIV infection
– Possibly stress
– Probably osteoporosis

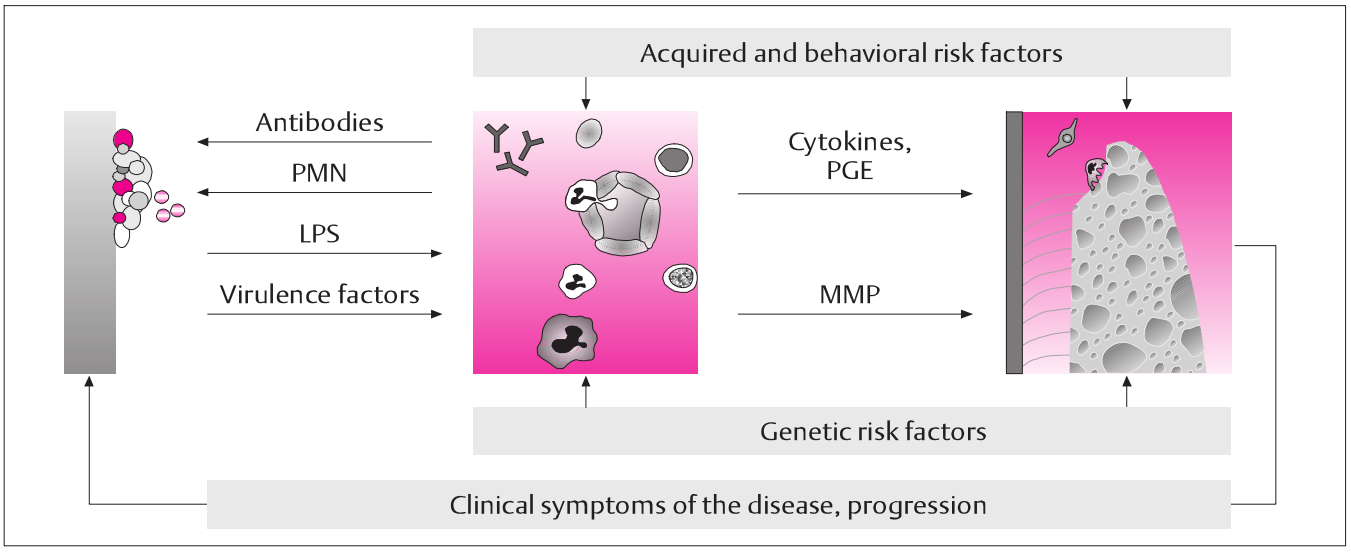


Fig. 3.**9** Current paradigm of the pathogenesis of periodontitis. Both the inflammatory and immunological host responses to bacterial challenge and the bone and connective tissue metabolism are controlled by genetic factors and modulated by acquired and behavioral risk factors. Bacteria and host are closely interrelated in the sense that clinical alterations following initiation and progression of periodontitis selectively support the growth of pathogenic bacteria. (Adapted from Page and Kornman 1997)

Table 3.**4** Possible immune defense anomalies in patients with aggressive periodontitis

Anomalies	Biological effects
Dysfunctions of neutrophilic granulocytes	• Reduced chemotaxis • Reduced number of receptors for FMLP, C5a, IL-8 • Mutation and defect FMLP receptor • Reduced capacities for phagocytosis and intracellular killing • Reduced release of leukotriene B • Enhanced release of superoxide anion
Protective subclass IgG2 antibodies	• Are rarely formed in generalized aggressive periodontitis
Polymorphisms in the FcγRIIa receptor	• Homozygous individuals with arginine instead of histidine at position 131 are probably at higher risk of infection with *A. actinomycetemcomitans*
Monocytes response to bacterial LPS	• Increased release of PGE_2 and IL-1
Imbalance of activities of different T cell subpopulations	• Suppressed Th1 response (IL-2, INF-γ, TNF) • Increased Th2 response (IL-4, IL-5, IL-6): polyclonal B cell stimulation

- Genetic factors:
  - In individuals of Northern European heritage, polymorphisms in the IL-1 gene cluster have been associated with chronic periodontitis.
  - A polymorphism of the FcγRIIa receptor (arginine instead of histidine at position 131) increases, in homozygous individuals, the susceptibility to infection with gram-negative, facultatively anaerobic pathogens including *A. actinomycetemcomitans*.
  - There is a weak association between polymorphisms of the FcγRIIIb receptor (NA1-NA2) and refractory chronic periodontitis.
  - A possibly genetic lack of granulocyte receptors for IL-8, C5a, and FMLP, or their dysfunction, has been associated with an increased risk for aggressive periodontitis in certain populations.
- Especially in cases of aggressive periodontitis, further immune defense anomalies have been identified (Table 3.**4**).

4 Epidemiology of Periodontal Diseases

Epidemiological Terminology

General Remarks

- Definitions:
  - *Incidence:* number of new cases of disease within a certain time period in relation to the number of individuals in the population examined; in cariology, the term *caries increment*, i. e., the number of new carious lesions within a given time period, is used.
  - *Prevalence:* number of diseased individuals at a certain point of time in relation to the number of individuals in the population examined.
  - *Lethality:* number of fatalities of a given disease within a certain time period in relation to the number of diseased individuals.
  - *Mortality:* number of fatalities of a given disease in relation to the number of individuals in the population examined; mortality = incidence × lethality.
- *Descriptive epidemiology* is occupied with:
  - the *distribution* of diseases in the population;
  - possible *etiological factors* associated with health, disease, defects, handicaps, and fatalities.
- Regularly conducted surveys may allow:
  - trends and developments to be uncovered;
  - new hypotheses to be formulated.
- *Analytical epidemiology* attempts to verify hypotheses.
  - Descriptive *cross-sectional studies* are generally unsuited to identifying risk factors. However, they may allow the association between a disease and a particular (potential risk) factor to be calculated.
  - The same applies for retrospective *case–control studies*, in which cases of diseased individuals are matched with healthy controls.
  - Particularly important are *prospective cohort studies*, which allow the identification of existing potential risk factors.
- *Risk factors* are associated with the development and progression of the disease, but are not necessarily causative.
- In order to confirm a risk factor as part of the causal chain, Hill's criteria (see Chapter 2) are usually quoted:
  - *Strength of association* between factor and disease.
    - One possible measure is the relative risk, which is given as the factor by which the likelihood of getting the disease on exposure to the agent is increased.
    - A serious association is taken to exist if there is a relative risk of at least 2.
  - *Consistency of association* in different populations:
    - Association in the same direction.
    - No overt discrepancies in its strength.
  - *Temporal sequence:* the factor must precede outbreak of the disease (principle of cause and effect).
  - *Specificity of association:* causative factors should generally be associated with only one disease. However, lack of specificity is not a reason to reject causality.
  - *Dose–response effect.*
  - *Biological plausibility.*
  - *Experimental evidence:*
    - Definitive confirmation in interventional studies, e. g., randomized controlled trials.
    - However, elimination of the risk factor does not always lead to healing.
- Findings from epidemiological investigations have a large impact on the planning and conduct of public health campaigns at the population level.

Periodontal Epidemiology

- Specific problems arise in periodontology:
  - Inflammatory periodontal diseases are almost ubiquitous (very high prevalence)
  - Progression of the disease is still not fully understood:

  - Lesions usually develop very slowly.
  - There is some evidence that progression is discontinuous.
- Loss of tooth-supporting apparatus is essentially irreversible.
- Lack of a pathognomonic feature. It is not entirely clear whether different expressions of the disease represent different entities.
- Complex, multifactorial etiology.

➤ In epidemiological studies on periodontal health in a given population, three different parameters have to be assessed:
- *Prevalence* of the disease, i.e., the proportion of diseased individuals
- *Extent* of the disease, i.e., the number or proportion of affected subunits (teeth, tooth surfaces)
- *Severity* of the disease: amount of attachment loss, depth of periodontal pockets

➤ These fundamentals were not observed in investigations conducted before about 1975.
- Data from older studies cannot therefore be compared with data from more recent surveys.
- In older studies, tooth loss in particular was erroneously considered to be definitive evidence of severe periodontal disease.

➤ Recent evidence from the 1980s and 1990s has profoundly modified our understanding of the natural history of periodontitis:
- Gingivitis does not lead inevitably to periodontitis.
- Aggressive forms of periodontitis, which result in premature tooth loss, are quite rare.
- Mild or moderate forms are widespread. Almost every adult shows some loss of attachment without experiencing any functional problems.
- Even after age of 35 years, periodontitis is not the main cause of tooth loss.

Examination Methods

General Remarks

➤ The extent and the severity of periodontal diseases and etiological factors such as plaque and dental calculus are often assessed as qualitative (ordinal) variables by means of index systems. Several problems inherent in this approach must be considered:
- A subjective element in data collection.
- Intermediate stages are not defined.
- Opinions often differ about the best (or valid) method of statistical analysis.

Table 4.1 Gingival Index (Löe and Silness 1963. Not in reference list.)

Score	Description
0	Normal gingiva
1	Slight inflammation • Slight change in color • Slight edema • No bleeding after probing of the sulcus
2	Moderate inflammation • Redness • Edematous swelling • Glazy appearance, loss of stippling • Bleeding after probing of the sulcus
3	Severe inflammation • Distinct redness • Distinct edematous swelling • Ulceration • Tendency to spontaneous bleeding

Assessment of Gingival Inflammation

➤ Indices for assessment of the degree of severity of gingivitis:
- *PMA Index:* papilla, marginal and attached gingiva are assessed separately; up to five degrees of severity each (Massler 1967)
- *Gingival Index* (GI, Löe and Silness 1963), mainly used in scientific studies (Table 4.1)
- *Sulcus Bleeding Index* (SBI, Mühlemann and Son 1971)
- *Papilla Bleeding Index* (PBI, Saxer and Mühlemann 1975) (Table 4.2).

Table 4.2 Papilla Bleeding Index (Saxer and Mühlemann 1975. Not in reference list.)

Score	Description of bleeding after cautious probing the sulcus
0	No bleeding
1	Bleeding point
2	Several isolated bleeding points or small bleeding area
3	Interdental triangle fills with blood
4	Profuse bleeding

- In some index systems, the sulcus is cautiously probed and the proportion of bleeding gingival units calculated: *Gingival Bleeding Index* (Ainamo and Bay 1975).
- An especially simple diagnostic finding is presence or absence of bleeding upon probing to the base of the sulcus or pocket.

Bacterial Deposits

- Index systems for plaque and dental calculus:
  - *Oral Hygiene Index* (OHI or OHI-S; Greene and Vermillion 1960, 1964). May be subdivided into:
    - ODI: debris index (soft deposits);
    - OCI: calculus index;
  - OHI-S (simplified): considers labial surfaces of FDI teeth nos. 16, 11, 26, and 31, as well as lingual surfaces of teeth 36 and 46. Grading: deposits cover up to one-third of the tooth surface (grade 1), more than one-third (grade 2), or more than two-thirds (grade 3).
  - *Quigley-Hein Index* (QHI): assessment of plaque on the labial surfaces of the anterior teeth, with grading from 0 to 5 (Quigley and Hein 1962).
  - *Turesky modification of QHI:* assessment of facial and lingual surfaces of all teeth (Turesky et al. 1970).
  - *Plaque Index* (PlI; Silness and Löe 1964): assessment of marginal plaque (Table 4.**3**).
- Simple systems take account only of the presence or absence of visible plaque and calculate the percentage of positive surfaces:
  - *Plaque Control Record* (PCR; O'Leary et al. 1972), assesses four sites on each tooth present.

Table 4.**3** Plaque Index (Silness and Löe 1964. Not in reference list.)

Score	Description
0	No plaque
1	Thin film of plaque which is invisible to the naked eye and may be noticed only by running a probe along the tooth surface
2	Moderate accumulation of plaque which is clearly visible
3	Abundance of plaque

Combined Indices

- Systems combining the assessment of gingivitis, periodontitis, and even etiological factors in just one index were very popular, especially in the middle of the last century:
  - *Periodontal Index* (PI; Russel 1967); no special instruments are necessary:
    - 1 or 2: localized or circumferential gingivitis, respectively
    - 6: initial periodontitis without impairment of function
    - 8: advanced periodontitis with functional impairment
  - *Periodontal Disease Index* (PDI; Ramfjord 1959):
    - 1–3: severity of gingivitis
    - 4–6: attachment losses of up to 3 mm, 4–6 mm, and 7 mm or more.
- *Community Periodontal Index* (CPI; Ainamo et al. 1982, World Health Organization 1997):
  - Assessment of the highest score in each sextant (Table 4.**4**)
  - May also be used for individual periodontal screening and recording (PSR).

Attachment Loss

- The amount of supporting tissue which has actually been lost can be measured:
  - Clinical attachment loss:
    - Vertical distance between the clinically probed bottom of the pocket or sulcus and the cementoenamel junction.

Table 4.**4** Community Periodontal Index for Treatment Needs (CPITN; Ainamo et al. 1982), now known as the Community Periodontal Index (CPI; WHO 1997). It may be also used for individual periodontal screening and recording (PSR)

Score	Description
0	No bleeding after probing with a special WHO probe
1	Bleeding after probing
2	Supra- or subgingival calculus or insufficient restoration margins, probing depth less than 4 mm
3	Probing depth 4–5.5 mm
4	Probing depth of 6 mm or more

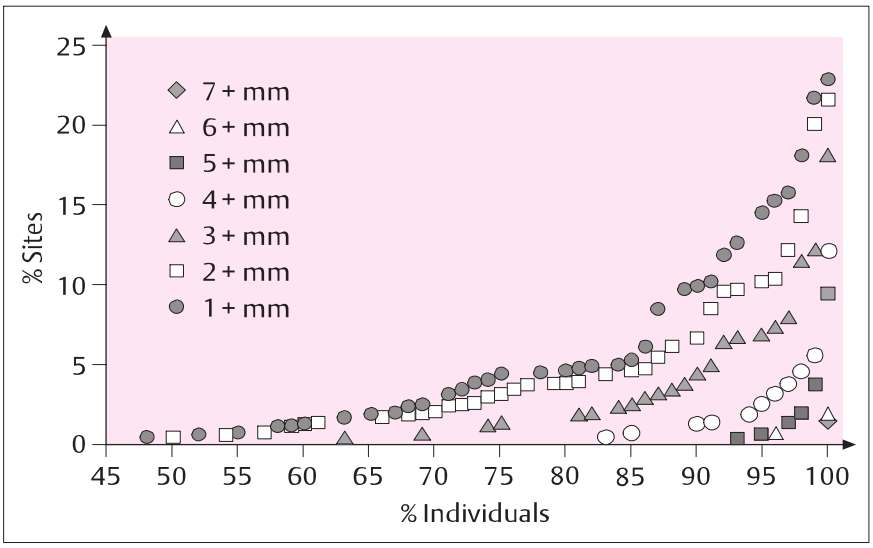


Fig. 4.1 Percentile plot of cumulative frequencies of attachment loss in a group of young adults up to 30 years of age. Average attachment loss was 0.08 ± 0.14 mm. Multivariate representation (prevalence, extent, and severity of the disease) allows almost all of the information to be preserved. Example: attachment losses were observed in 53 % of the examined population; 38 % had at least one site with attachment loss of 3 mm or more, 18 % at least one site with attachment loss of 4 mm or more. The proportion of individuals with a notable number of sites (e. g., more than 5 %) with relevant attachment loss (3 mm or more) was about 9 %. One individual had one site with 7 mm attachment loss

- If the cementoenamel junction is located subgingivally, the periodontal probing depth is determined first, then the cementoenamel junction is identified with the probe and the distance to the gingival margin subtracted from the probing depth.
- Horizontal attachment loss is usually related to furcation involvement.

➤ An index that simultaneously assesses the extent and severity of periodontal destruction is the *Extent and Severity Index* (ESI; Carlos et al. 1986), giving a bivariate measure as follows:
- First value: disease extent, i. e., percentage of teeth or tooth surfaces involved for a defined threshold value, e. g., 3 mm.
- Second value: disease severity, i. e., average attachment loss at sites lying above the threshold.
- Example: an ESI of 3 : 60; 3.5 means that 60 % of teeth or sites have attachment loss of 3 mm or more, and the average attachment loss of these teeth or sites is 3.5 mm.

➤ The information is largely preserved in the bivariate representation of cumulative frequencies of probe parameters in the form of percentile plots (Fig. **4.1**).

Epidemiology of Plaque-Induced Periodontal Diseases

General Remarks

➤ Inflammatory periodontal diseases associated with dental plaque are among the most frequent diseases of man.
- Inflammatory alterations of the gingiva are found in most children even in the primary dentition.
- The prevalence and extent of gingivitis reach their maximum at the onset of puberty, at about 11 years in girls, in boys about 2 years later (Fig. **4.2**).
- On the other hand, the severity of gingivitis does not increase during puberty.

➤ The prevalence of periodontitis had been studied all over the world since the 1950s.
- Examination criteria have differed considerably.
- In most studies, only prevalence of the disease was determined (Fig. **4.3**):

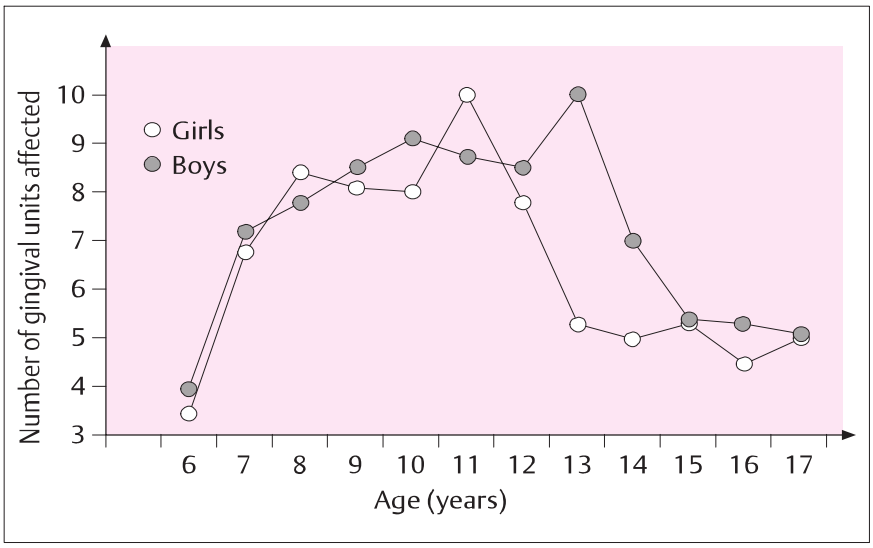


Fig. 4.**2** Extent of gingivitis (PMA index: papilla, marginal gingiva, attached gingiva) in children (Massler et al. 1952). A maximum is reached at the onset of puberty, in girls at age 11, in boys at age 13

- Gingivitis seems to diminish continuously after the age of 15 years.
- Concomitantly, periodontitis increases dramatically, reaching 100 % after the age of 35 years.
- Tooth loss gradually affects more and more individuals.

Natural History

➤ The natural history of periodontitis was studied in a longitudinal investigation of Sri Lankan tea workers who had received no dental care (Fig. 4.**4**):
- 8 % of the population developed a rather aggressive form of the disease:
  - Virtually all teeth had been lost by the age of 40 years.

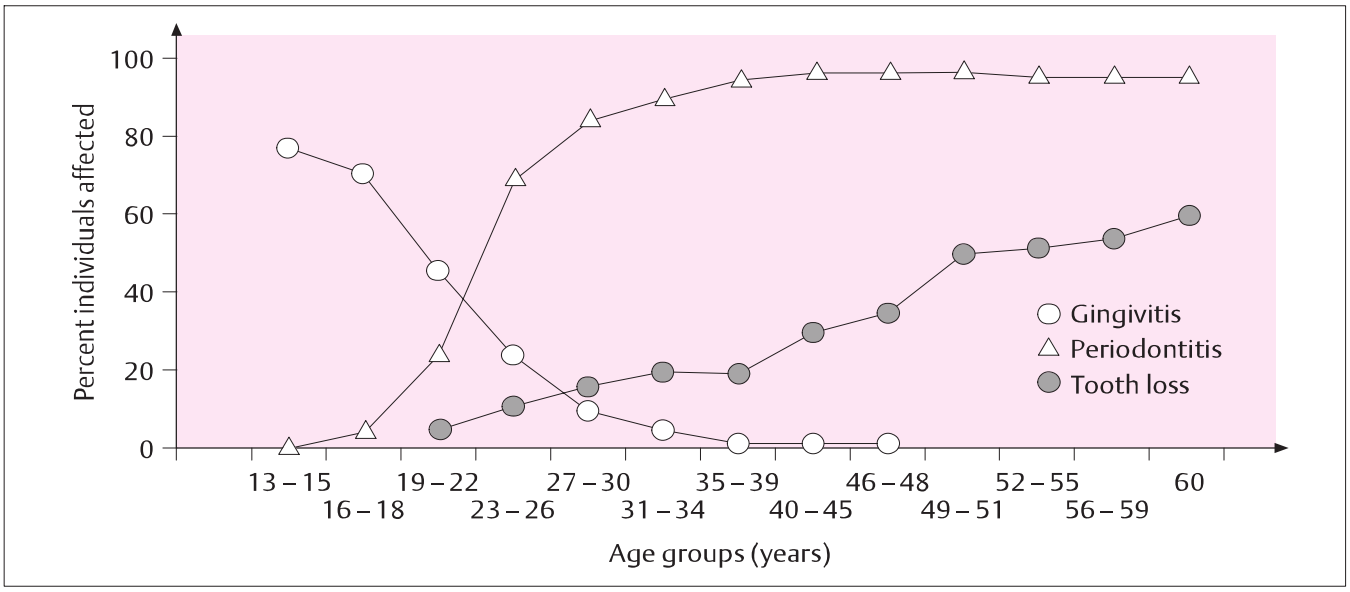


Fig. 4.**3** Prevalence of gingivitis, periodontitis, and tooth loss (Marshall-Day et al. 1955). In adolescents, a very high prevalence of gingivitis was noted, which subsequently rapidly declined. Simultaneously, however, the prevalence of periodontitis increased steeply, reaching almost 100 % after the age of 35 years. The proportion of individuals with tooth loss increased linearly from the age of about 20 years onwards

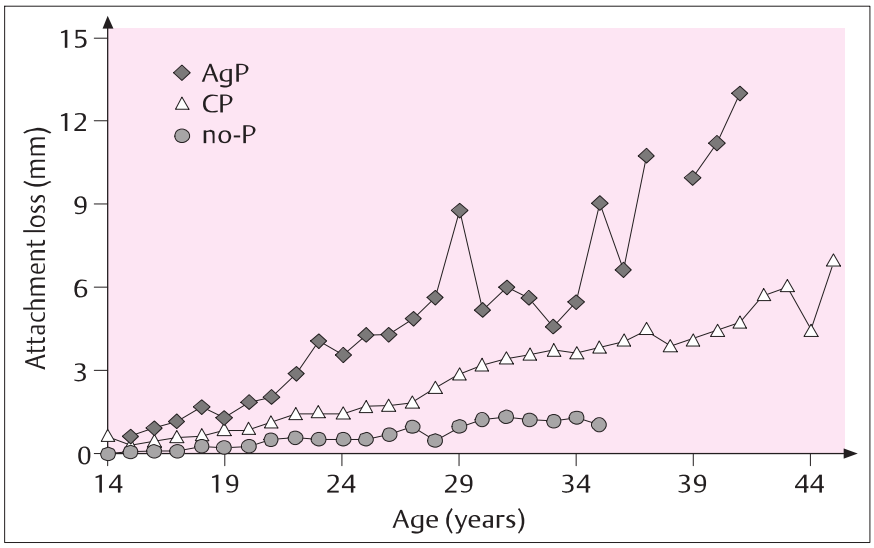


Fig. 4.**4** Average age-dependent attachment loss in Sri Lankan tea workers, who were studied for 15 years (Löe et al. 1986). Eight percent developed an aggressive form of periodontitis (AgP) and 81 % a chronic form (CP), while in 11 % no periodontitis was diagnosed (no-P)

- Average attachment loss was more than 1 mm per year.

– 81 % of the population developed a mild, chronic, form of periodontitis:
- Any tooth loss did not impair the function of the dentition.
- Annual attachment loss increased from an average of 0.3 mm by the age of 30 to 0.5 mm at the age of about 45.

– In 11 % of the population no relevant attachment loss was observed.

➤ *Note:* This and other investigations have shown that about 7–15 % of a given population suffer from a severe form of periodontitis which leads to premature tooth loss.

➤ The progression rates of periodontitis have a basically normal distribution.

NHANES III

➤ The Third National Health and Nutrition Examination Survey (NHANES III) is the largest population-based study carried out so far in the USA; it was conducted between 1988 and 1994 (Figs. 4.**5** and 4.**6**):

– Attachment losses of 3 mm or more were found in 53 % of 30- to 90-year-olds.

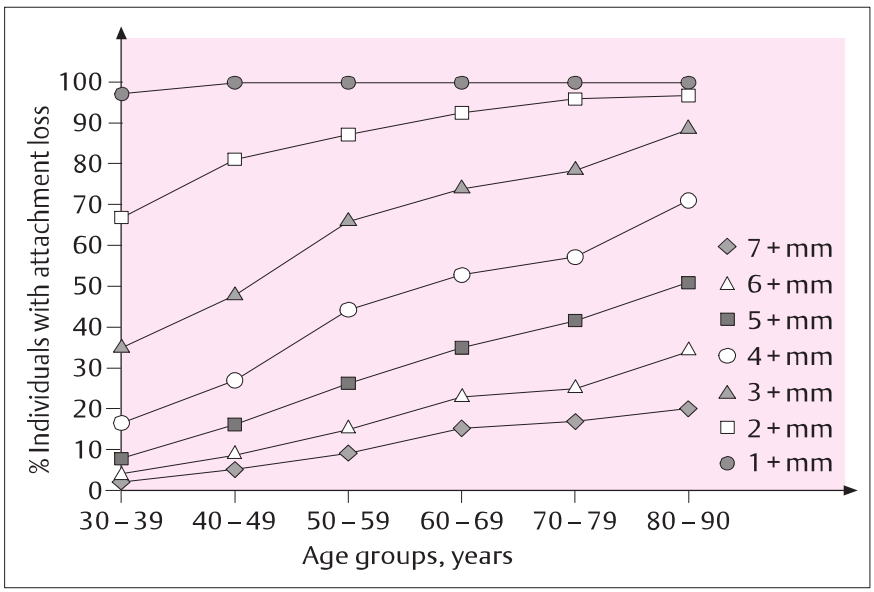


Fig. 4.**5** NHANES III. Prevalence of individuals with attachment loss. (Adapted from Albandar et al. 1999)

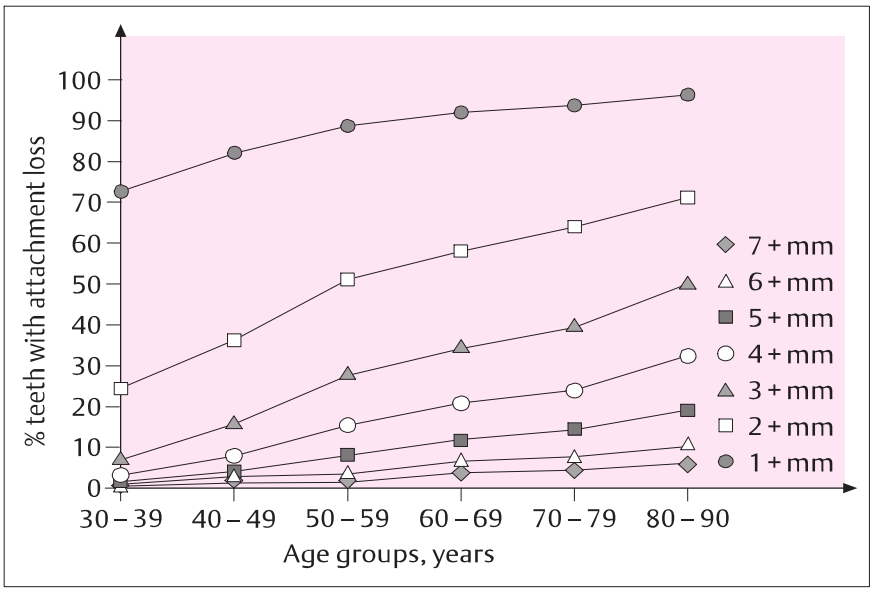


Fig. 4.6 NHANES III. Extent of attachment loss (percentage of teeth affected). (Adapted from Albandar et al. 1999)

- In each individual about 20% of the teeth were affected, on average.
- Probing depths of 3 mm or more were observed in about 64% of individuals.
- On average, 30% of teeth were affected.

➤ Two random quadrants (one in the maxilla, one in the mandible) were examined, with periodontal probing of the mesiobuccal and midbuccal surfaces of all teeth excluding third molars.

➤ Among dentate individuals with six or more teeth, different degrees of severity were defined as follows:

- Advanced periodontitis:
  - 2 or more teeth (or ≥ 30% of the teeth examined) with a probing depth of 5 mm or more, or
  - 4 or more teeth (or ≥ 60% of the teeth examined) with a probing depth of 4 mm or more, or
  - 1 or more multirooted teeth with through-and-through furcation involvement
- Moderate periodontitis:
  - 1 tooth with a probing depth of 5 mm or more, or
  - 2 or more teeth (or ≥ 30% of the teeth examined) with a probing depth of 4 mm or more, or
  - 1 or more multirooted teeth with partial furcation involvement (not through-and-through)
- Mild periodontitis:
  - 1 or more teeth with a probing depth of 3 mm or more, or
  - 1 or more multirooted teeth with partial furcation involvement (not through-and-through)

➤ This classification produced the following results:

- At least 35% of the dentate US population between 30 and 90 years of age has periodontitis, 22% a mild form, and 13% a moderately advanced or advanced form.
- The prevalence and extent of attachment loss increase with age. In the highest age group, the prevalence of periodontitis decreases because of tooth loss and recession.
- Severe forms of periodontitis affect more men than women. More African Americans and Hispanics are affected than whites.

➤ For the first time, furcation involvement was assessed in a population sample:

- About 14% of individuals aged 30 years and older had at least one tooth with furcation involvement.
- On average, 7% of multirooted teeth were affected in each individual.
- A single furcation involvement was observed in 10%, while 4% had 2 or more teeth affected.

Europe

- A meta-analysis of 47 Community Periodontal Index of Treatment Needs (CPITN) studies from 24 countries conducted between 1982 and 1992 showed that:
  - The weighted mean prevalence of shallow pockets (CPITN 3) is 36% in Western European countries and 45% in Eastern Europe.
  - The weighted mean prevalence of deep pockets (CPITN 4) is 9% in Western European countries and 23% in Eastern Europe.
  - The average number of sextants affected by periodontitis (extent) was generally low: 0.2–2.4 for CPITN 3, 0–0.8 for CPITN 4.

Early-Onset (Aggressive) Periodontitis

- The largest population-based study of 14- to 17-years-olds was conducted by the National Institute of Dental and Craniofacial Research (NIDCR) in 1986–1987 in the USA.
  - About 0.5% of individuals had localized aggressive periodontitis: 0.1% among whites and 2% among African Americans.
  - 0.1% had generalized aggressive periodontitis: 0.03% of whites and 0.6% of African Americans.
- Worldwide, the prevalence of aggressive periodontitis seems to vary considerably (Fig. **4.7**). *Note:* the disease is apparently more prevalent in black populations:
  - 0.1–0.2% in Europe
  - More than 6% in Uganda
  - 0.8% in Nigeria
  - 3.7% in Brazil

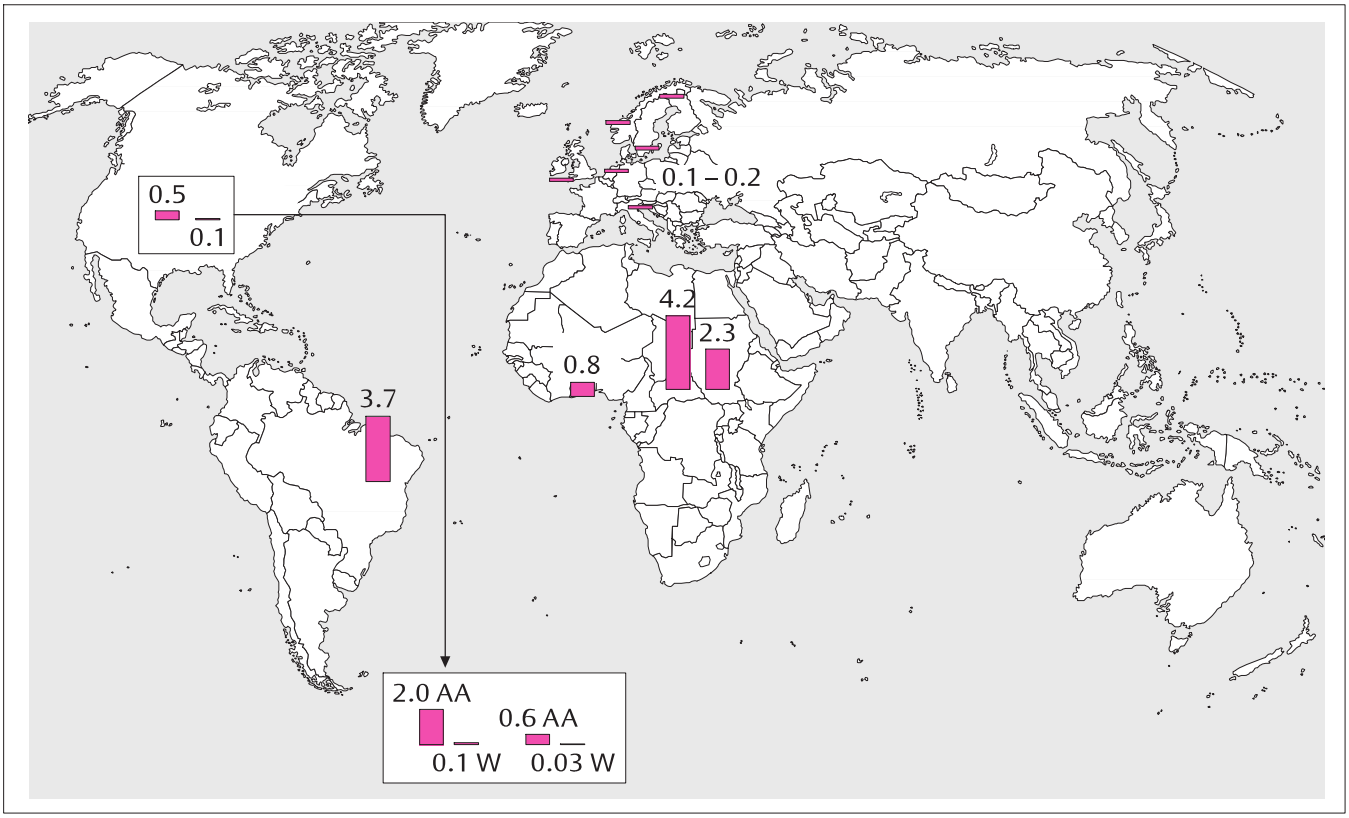


Fig. 4.7 Worldwide distribution of aggressive periodontitis in children and adolescents (in %). Data from the USA distinguish between localized aggressive periodontitis (about 0.5%, 2% in African Americans (AA), 0.1% in whites (W) and generalized aggressive periodontitis (about 0.1%, 0.6% in African Americans, 0.03% in whites) in 14- to 17-year-olds. Recently, in Uganda respectively 4.2 and 2.3% of adolescents were found to be affected by localized and generalized aggressive periodontitis. (Adapted from Löe and Brown 1991, supplemented by recent data)

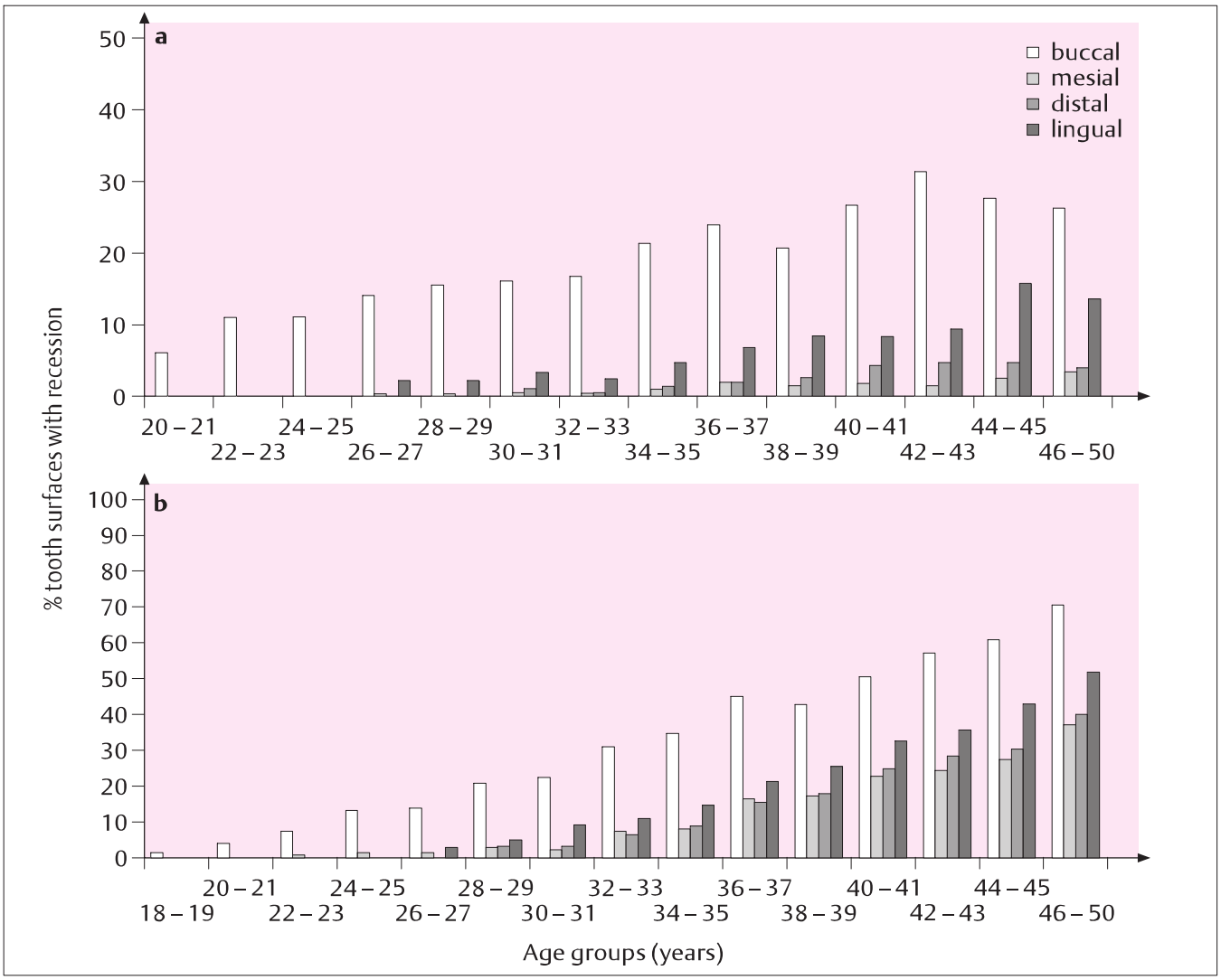


Fig. 4.8 There are two different forms of gingival recession.

a Age-dependent increase of the proportion of tooth surfaces with recession in a Norwegian population with excellent dental service and a high level of oral hygiene. Buccal and, to a lesser extent, lingual surfaces are those most affected.

b Age-dependent increase in the proportion of tooth surfaces with recession in workers in a tea plantation in Sri Lanka with no dental service and no special oral hygiene measures. In these individuals, recession extent increases also on proximal surfaces. (Adapted from Löe et al. 1992)

Prevalence, Extent, and Severity of Gingival Recession

- Gingival recession apparently occurs in two different forms (Fig. 4.8):
  - One form is mainly induced by *injury*, especially due to inappropriate tooth brushing. In this form, the gingiva is virtually free of inflammation:
    - Most often, buccal surfaces of canines and premolars are affected.
    - Less often, lingual or palatal surfaces—usually the first maxillary molars—are affected.
  - The other form is due to *longstanding chronic inflammation* in the course of periodontitis.
- In NHANES-III, the following observations were made:
  - 58% of those more than 30 years old had at least one site with ≥ 1 mm recession.
  - Recession of at least 3 mm was found in 22% of individuals.
  - On average, 22% of teeth were found to have a recession of at least 1 mm.

5 Prevention of Periodontal Disease

Prevention-Oriented Dentistry

General Remarks

➤ For many chronic diseases, the primary objective is *prevention*. Prevention of caries and periodontal disease is possible for the following reasons:
- – Both are *infectious diseases*. Suppression and control of the oral pathogens responsible for the disease process to a large extent appears possible.
- – *Health awareness* among the general public is increasing.
- – *Oral hygiene measures* are well established.
- – *Fluoridation* is available in several forms to most parts of the population.

➤ Preventive measures are traditionally divided into primary, secondary, and tertiary. To prevent caries and periodontal disease, the following may be considered:
- – Primary prevention:
  - • Improvement of individual oral hygiene
  - • Appropriate diet which should not harm dental health
  - • Local fluoridation
  - • Regular check-ups
- – Secondary prevention:
  - • Early assessment and treatment of any diseases in the oral cavity
  - • Professional tooth cleaning
- – Tertiary prevention:
  - • Systematic therapy
  - • Prevention of complications

Karlstad Study

➤ The paramount importance of a consistently carried out preventive program for the establishment of oral health was proved in the Karlstad study (Axelsson et al. 1991):
- – Test group: 375 adult patients who were offered the following:
  - • Oral hygiene training.
  - • Professional tooth cleaning.
  - • Conventional caries treatment.
  - • Recall every 2nd month for the first 2 years, thereafter every 3rd month, with reinforcement of oral hygiene and fluoridation.
- – Control group: 180 patients who received standard dental care:
  - • Oral hygiene training.
  - • Professional tooth cleaning.
  - • Conventional caries treatment.
  - • Thereafter, patients were referred back to their original dentist with check-ups once a year.
- – This controlled study had to be stopped for ethical reasons after 6 years:
  - • On average, a further 12–15 surfaces became carious in the control group, compared to only 0–1 surfaces in the test group.
  - • Attachment loss in the control group amounted to 0.7–1.5 mm. On average, subjects in the test group showed some attachment gain (Fig. 5.**1**).
  - • After 6 years, therefore, the control patients were offered the same care as the test group patients.

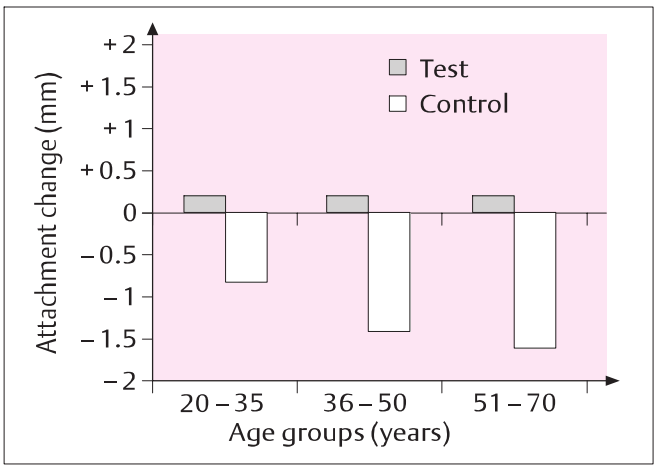


Fig. 5.**1** During the first six years of the Karlstad study, attachment losses occurred only in the control group (yearly visits to the dentist). (Adapted from Axelsson et al. 1991)

- After 6 years, an individual schedule for maintenance visits was established in test patients; 95% of the study participants needed only 1–2 prophylaxis sessions per year.

➤ During the whole study period of 15 years, only 59 teeth were lost in the 375 patients comprising the test group.

➤ The main reason for the generally stable conditions was a permanently low proportion of 20% or less of tooth surfaces with dental plaque.

➤ The main message of the Karlstad study is:
- The two most important diseases of the oral cavity, caries and periodontal disease, can be totally prevented.
- On the other hand, at a population level, this can only be achieved by an enormous, probably disproportionate effort.

Possibilities for Prevention

➤ During the past 10–15 years the general view of periodontitis in the population at large has been thoroughly revised:
- Susceptibility to the disease differs considerably among individuals. Not every form of gingivitis inevitably develops into destructive periodontitis
- The prevalence of aggressive forms of the disease in particular is lower than was thought for a long time; it is possible that it will further decrease with time. Many people keep their full dentitions despite having gingivitis or mild forms of periodontitis.
- Whether the risk group of periodontally susceptible individuals will benefit from further improvement of oral health in general is doubtful.

➤ This reassessment has important consequences for public health:
- Prevention of the early stages of gingivitis at the population level is an unrealistic aim and probably unnecessary.
- Although the dogma of the causal chain "plaque → gingivitis → periodontitis" can be found in most contemporary textbooks, the current concept of the relative importance of dental plaque for pathological conditions in the oral cavity is changing
- Instead of total plaque control, what is aimed at is a considerable reduction in the *proportion of oral pathogens* in the complex dental microbiota, via changes in the ecological conditions.
- Rather than planning periodontal surgery for the mass of the population, one of the most important tasks is intensive diagnostic, preventive, and therapeutic care for a small group of susceptible patients.

➤ Large parts of the population may be managed without periodontal surgery by taking a more conservative approach to periodontal therapy (secondary prevention):
- These measures require detailed information to be given on the etiology of periodontal diseases and how to prevent them (motivate the patient).
- Before any definitive dental treatment commences, a short course (two to three sessions) of oral hygiene instruction and prophylaxis should be performed.
- A conscious distinction should be made between patients who visit a dentist only sporadically when they have an acute complaint and the relatively small group of patients who desire systematic therapy, i.e., comprehensive care.
- With few exceptions (e.g., traumatic injury, handicapped patients), *no definitive treatment* should be given unless relevant preventive measures have achieved some measurable success.
- In particular, restoring patients' ability to practice appropriate oral hygiene is mandatory.
- In most patients, professional tooth cleaning will need to be repeated only at relatively long intervals, e.g., every 6–12 months.

➤ *Note:* Continuous nonsurgical periodontal therapy reduces the rate of tooth loss by more than 50%.

Measures at the Population Level

➤ *Strategies affecting the whole population.* At the population level, even quite small improvements in oral hygiene can have a dramatic impact on periodontal health in general. To achieve success at this level, information about factors that interfere with improvement of oral hygiene are needed:
- Educational measures and campaigns for oral health improvement should be imple-

mented within the context of established *general health education:*
  - The starting point should be individual need, not objective deficiency.
  - Basic information should be provided about plaque, mechanisms of prevention, the possibility of treatment, the efficacy of commercial products, etc.
  - Enable rather than prescribe.
  - Less professional intervention, more self-help.
  - Make healthier alternatives easier.
- Health education at various levels. Kindergarten nurses, teachers, pediatricians, and the whole dental team promote oral hygiene within the framework of general education programs in:
  - General cleanliness and body care
  - Disease prevention
  - Improved self-esteem
- All health care workers participating in these programs should receive training in the professional teaching of effective oral hygiene.
- Oral hygiene measures should be possible in schools and in the workplace, and should be regarded as a matter of course:
  - Sanitary installations should be accessible to all.
  - Supply of appropriate remedies
  - Differing age groups and levels of education should be taken into account.

Secondary and Tertiary Prevention

➤ Secondary and tertiary prevention in the context of comprehensive treatment:
- *Secondary preventive measures* are indicated whenever *comprehensive treatment* of all pathologic conditions in the oral cavity and oral rehabilitation is planned.
- For most periodontally diseased patients, simple measures are sufficient for long-term maintenance of the dentition:
  - Training in effective oral hygiene.
  - Regular supra- and subgingival scaling
  - Periodontal surgery is indicated in only a few cases and situations
- A central aspect of treatment planning is to consider the *strategic importance* of individual teeth.
  - The extraction of any periodontally diseased tooth from an intact dental arch is a matter of great responsibility. Considerable costs and risks may be associated with prosthetic rehabilitation.
  - Greater therapeutic efforts should be made in the case of strategically important teeth. An early referral to a periodontal specialist is desirable.
  - On the other hand, early extraction of diseased, strategically irrelevant teeth may be indicated in order to prevent costly and risky prosthetic constructions (e.g., periodontally diseased wisdom teeth and second molars).

Preferential Treatment of High-Risk Groups

➤ Patients in high-risk groups should be identified and given priority:
- A fairly small group of patients (not more than 15 % of the population) is responsible for virtually all tooth loss due to periodontitis:
  - General risk factors such as smoking, diabetes mellitus, and genetic factors such as a specific IL-1 genotype, etc., are now well established.
  - The presence of a number of periodontal pathogens such as *A. actinomycetemcomitans*, *P. gingivalis*, and *T. forsythia* seems to increase the risk of active periodontitis in certain individuals.
- *Note:* High-risk patients incur considerable diagnostic and therapeutic costs:
  - Microbiological and, in some cases, genetic tests
  - Periodontal surgery, including regenerative measures
  - Adjunctive antibiotic therapy
  - Expensive epithetic and/or prosthetic constructions
  - Oral implants

➤ *Note:* Periodontally diseased patients in a high-risk group should preferably be treated by periodontal specialists.

6 Classification of Periodontal Diseases

Current Classification

General Remarks

➤ Classification systems provide a framework within which the etiology, pathogenesis, diagnosis, and therapy of diseases can be scientifically studied. They enable the clinician to assess individual treatment needs.

➤ The current classification scheme of periodontal diseases is based on the consensus reports of the International Workshop for a Classification of Periodontal Diseases and Conditions held in 1999. The most important results of this workshop were:

– Delineation of detailed criteria for classification of gingival diseases.

– A new classification of periodontitis. The previous classification according to age of onset and progression rate was dismissed:
  • The term "adult periodontitis" was dropped. Most of these cases are characterized by slow disease progression; thus, the term "chronic periodontitis" may be more appropriate.
  • The term "early-onset periodontitis" was dropped. This heterogeneous group previously grouped prepubertal and juvenile periodontitis together. Many of these cases are characterized by rapid progression and are more appropriately termed "aggressive periodontitis."
  • Some cases of chronic periodontitis are found also in children and adolescents.

Table 6.1 Classification of periodontal diseases and conditions

I Gingival diseases

A Dental plaque-induced gingival diseases

1 Gingivitis associated with dental plaque only
- a Without other local contributing factors
- b With local contributing factors

2 Gingival diseases modified by systemic factors
- a Associated with the endocrine system
  - 1) Puberty-associated gingivitis
  - 2) Menstrual cycle-associated gingivitis
  - 3) Pregnancy-associated
    - a) Gingivitis
    - b) Pyogenic granuloma
  - 4) Diabetes mellitus-associated gingivitis
- b Associated with blood dyscrasias
  - 1) Leukemia-associated gingivitis
  - 2) Other

3 Gingival diseases modified by medications
- a Drug-influenced gingival diseases
  - 1) Drug-influenced gingival enlargements
  - 2) Drug-influenced gingivitis
    - a) Oral contraceptive-associated gingivitis
    - b) Other

4 Gingival diseases modified by malnutrition
- a Ascorbic-acid-deficiency gingivitis
- b Other

B Non-plaque-induced gingival lesions

1 Gingival diseases of specific bacterial origin
- a *Neisseria gonorrhoea*-associated lesions
- b *Treponema pallidum*-associated lesions
- c Streptococcal species-associated lesions
- d Other

2 Gingival diseases of viral origin
- a Herpesvirus infections
  - 1) Primary herpetic gingivostomatitis
  - 2) Recurrent oral herpes
  - 3) Varicella-zoster infections
- b Other

3 Gingival diseases of fungal origin
- a *Candida* spp. infections
  - 1) Generalized gingival candidosis
  - 2) Linear gingival erythema
- b Histoplasmosis
- c Other

4 Gingival lesions of genetic origin
- a Hereditary gingival fibromatosis
- b Other

Table 6.1 Continued

I Gingival diseases

B Non-plaque-induced gingival lesions

5 Gingival manifestations of systemic conditions
- a Mucocutaneous disorders
  - 1) Lichen planus
  - 2) Pemphigoid
  - 3) Pemphigus vulgaris
  - 4) Erythema multiforme
  - 5) Lupus erythematosus
  - 6) Drug-induced
  - 7) Other
- b Allergic reactions
  - 1) Dental restorative materials
    - a) Mercury
    - b) Nickel
    - c) Acrylic
    - d) Other
  - 2) Reactions attributable to
    - a) Toothpastes/dentifrices
    - b) Mouthrinses/mouthwashes
    - c) Chewing gum additives
    - d) Foods and additives
    - e) Other

6 Traumatic lesions (factitious, iatrogenic, accidental)
- a Chemical injury
- b Mechanical injury
- c Thermal injury

7 Foreign body reactions

8 Not otherwise specified

II Chronic periodontitis

A Localized
B Generalized

III Aggressive periodontitis

A Localized
B Generalized

IV Periodontitis as a manifestation of systemic disease

A Associated with hematological disorders

1 Acquired neutropenia
2 Leukemias
3 Other

B Associated with genetic disorders

1 Familial and cyclic neutropenia
2 Down syndrome
3 Leukocyte adhesion deficiency syndromes
4 Papillon–Lefèvre syndrome
5 Chediak–Higashi syndrome
6 Histiocytosis syndromes
7 Glycogen storage disease
8 Infantile genetic agranulocytosis
9 Cohen syndrome
10 Ehlers–Danlos syndrome (types IV and VIII)
11 Hypophosphatasia
12 Other

C Not otherwise specified

V Necrotizing periodontal diseases

A Necrotizing ulcerative gingivitis
B Necrotizing ulcerative periodontitis

VI Abscesses of the periodontium

A Gingival abscess
B Periodontal abscess
C Pericoronal abscess

VII Periodontitis associated with endodontic lesions

A Combined periodontic-endodontic lesions

VIII Developmental or acquired deformities and conditions

A Localized tooth-related factors that modify or predispose to plaque-induced gingival disease/periodontitis

1 Tooth anatomy
2 Dental restorations/appliances
3 Root fractures
4 Cervical root resorption and cemental tears

B Mucogingival deformities and conditions around teeth

1 Gingival/soft tissue recession
- a Facial or lingual surfaces
- b Interproximal (papillary)

2 Lack of keratinized gingiva
3 Decreased vestibular depth
4 Aberrant frenum/muscle position
5 Gingival excess
- a Pseudopocket
- b Inconsistent gingival margin
- c Excessive gingival display
- d Gingival enlargement (see IA3 and IB4)

6 Abnormal color

C Mucogingival deformities and conditions on edentulous ridges

1 Vertical and/or horizontal ridge deficiency
2 Lack of gingival/keratinized tissue
3 Gingival/soft tissue enlargement
4 Aberrant frenum/muscle position
5 Decreased vestibular depth
6 Abnormal color

D Occlusal trauma

1 Primary occlusal trauma
2 Secondary occlusal trauma

- Aggressive periodontitis may also develop at an older age. Many cases previously assigned to "refractory periodontitis" might be included in the category of aggressive periodontitis.

Plaque-Induced Gingival Diseases

General Remarks

➤ Plaque-induced gingivitis may occur on a periodontium without attachment loss/bone loss or on a periodontium with attachment loss/bone loss that is considered to be non-progressive. Characteristics are:
- Dental plaque in the region of the gingival margin.
- Defined histological alterations.
- Signs of inflammation and symptoms are confined to the gingiva:
  - *Swelling* due to edema or fibrosis
  - Red or bluish-red *change in color*
  - *Increased temperature* in the sulcus
  - Increased flow rate of *gingival exudate*.
- Inflammatory alterations are completely reversible after removal of the causative plaque.
- Local factors may influence the clinical picture of gingivitis, e.g., insufficient restorations, crowded teeth, etc.

Systemically Modified Gingival Diseases

➤ Enhancement of inflammatory responses to dental plaque due to *sex steroid hormones:*
- During puberty (Tanner stage 2 or higher):
  - In girls: estradiol levels (26 pmol/l)
  - In boys: testosterone levels (8.7 nmol/l)
- Immediately before ovulation during the menstrual cycle
- During pregnancy:
  - Mostly during the second and third trimester
  - Locally, a *pyogenic granuloma* may develop: exophytic granuloma of the gingiva, frequently involving an interdental papilla (pregnancy tumor).
- The increase of steroid hormones in gingival exudate during puberty and, in a much more pronounced manner, during pregnancy may result in:
  - distinct change of ecological conditions in the dentogingival region, leading to a possible advantage for, e.g., *P. intermedia*, *P. nigrescens*, and members of the *P. melaninogenica* group;
  - enhancement of inflammatory and immunological responses to microbial plaque due to the presence of specific receptors for sex steroid hormones in the gingiva.

➤ The inflammatory reaction to dental plaque is enhanced in individuals with poorly controlled *diabetes mellitus*, in particular in children with type I diabetes mellitus.

➤ In *acute leukemias* inflammatory reactions may lead to necrosis and/or gingival enlargement.

Drug-Influenced Gingival Diseases

➤ Drug-induced *gingival enlargements* have a genetic predisposition. Lesions are more pronounced at the anterior teeth, especially the interdental papillae. Young persons are more frequently affected. The following drugs may lead to gingival enlargement:
- *Anticonvulsants*, e.g., phenytoin:
  - Gingival enlargement in about 50% of patients.
  - Careful plaque control cannot prevent gingival enlargement but may reduce extent and severity.
- *Calcium channel blockers*, e.g., nifedipine, verapamil, diltiazem, etc.:
  - Frequently prescribed in patients aged 50 years or older for hypertension, arrhythmia, angina pectoris.
  - About 20% of patients develop gingival enlargement.
  - Oral hygiene may possibly influence the development of lesions.
- The *immunosuppressant* cyclosporine:
  - Following organ transplantation, or for the treatment of autoimmune diseases
  - 25–30% of patients develop gingival enlargement
  - Rigorous plaque control cannot prevent development of lesions but may reduce their extent and severity

- Combination therapy with calcium channel blocker has synergistic effects in regard to gingival enlargement.

– Inflammatory reactions to dental plaque may be enhanced by oral contraceptives.

Gingival Diseases Modified by Malnutrition

➤ As a result of malnutrition, immune defense mechanisms may be impaired, which may lead to an increased susceptibility to infections. In developed countries this is seen mostly in cases of:
– anorexia nervosa;
– chronic alcohol abuse.

➤ Chronic ascorbic acid (vitamin C) avitaminosis (scurvy) may enhance inflammatory reactions to dental plaque.

Gingival Diseases of Specific Bacterial Origin

➤ Specific infections with bacteria not usually resident in dental plaque:
– *Neisseria gonorrhoea:* usually asymptomatic enanthema
– *Treponema pallidum:* all three stages of syphilis may manifest in the oral cavity:
  - Primary affect: asymptomatic chancre with regional lymphadenopathy
  - Secondary affect: mucous patches
  - Tertiary affect: gumma, especially of the hard and soft palate
– Specific infections with β-hemolyzing streptococci
– Specific infections with other bacteria (e.g., mycobacterial infections)

Gingival Diseases of Viral Origin

➤ Apart from measles and rubella, a number of virus diseases manifest in the oral cavity, some of them also in the gingiva:
– Herpesviruses:
  - Herpes simplex virus (HSV-1: predominantly orally; HSV-2 predominantly anogenitally)
  - Varicella-zoster virus (human herpesvirus 3 [HHV-3])
  - Epstein–Barr virus (HHV-4)
  - Human cytomegalovirus (HHV-5)
– Papillomavirus

➤ Infections with herpesviruses usually exert acute symptoms:
– *Herpetic gingivostomatitis.* Primary infection with HSV-1 or HSV-2:
  - Highest incidence at the age of 2–4 years; occurrence in young adults is also possible.
  - Herpetic vesicles are found anywhere the oral cavity, with no predilection: lips, cheek, tongue, gingiva, and pharynx.
  - Vesicles have a high tendency of rupture leaving painful, fibrin-coated ulcers with diffuse erythema.
  - Fever, malaise, regional lymphadenitis.
  - Halitosis.
– *Recurrent oral herpes:* herpes labialis, cold sores. HSV-1 and HSV-2 persist in sensory ganglia.
  - Activation and local exacerbation may occur in cases of:
    - febrile common cold, UV radiation, menstruation, local stimuli, e.g., during dental treatment, emotional stress;
    - suppression or dysfunction of the immune system, radiotherapy.
  - In most cases there are local, highly contagious vesicles, for instance on the lip or at the gingiva.
– *Infection with varicella-zoster virus (HHV-3):*
  - Primary infection: chickenpox; virus persists in sensory dorsal root ganglia.
  - When the virus is activated (e.g., after UV radiation), lesions emerge within the area of the main sensory branch.
  - Virus activation can be the first indication of an unrelated, serious systemic disease.
– *Infection with Epstein–Barr virus (HHV-4):*
  - Cause of infectious mononucleosis.
  - Association with hairy leukoplakia on the lateral aspect of the tongue in HIV-seropositive patients
– *Infections with the human cytomegalovirus (HHV-5):* possible association with necrotizing ulcerative periodontal diseases.

Gingival Diseases of Fungal Origin

➤ Examples are aspergillosis, blastomycosis, candidosis, histoplasmosis, etc. The most frequent oral fungal infections are due to *Candida* spp., mostly *C. albicans.*

➤ *Candida* spp. are usually nonvirulent commensals of the oral cavity. Opportunistic infection may occur in immunologically suppressed patients, during therapy with antibiotics, glucocorticosteroids, antineoplastic drugs, in patients with poorly fitting dentures, etc.:
- *Primary oral candidosis:*
  - Acute pseudomembranous candidosis—white patches which may leave a red, bleeding surface when removed
  - Acute erythematous candidosis—diffuse erythematous areas
  - Chronic candidosis—pseudomembranous, erythematous, hyperplastic, nodular or plaque-like lesions
  - *Candida*-associated denture stomatitis, angular cheilitis, median rhomboid glossitis
  - Superinfected lesions (leukoplakia, oral lichen planus, lupus erythematosus)
- *Secondary oral candidosis* as a manifestation of systemic disease:
  - Diabetes mellitus.
  - Immune suppression. Refractory oral candidosis may be an early symptom of HIV infection. *Note:* Periodontal tissues may represent an entrance for systemic *Candida* infection in immune-suppressed patients.
  - A therapy-resistant *linear erythema* of the gingiva in HIV-seropositive patients may also be a *Candida* infection.

Gingival Manifestations of Mucocutaneous Diseases

➤ Some mucocutaneous diseases manifest in the oral cavity and also in particular in the gingiva, for example:
- Lichen planus
- Pemphigoid
- Pemphigus vulgaris
- Erythema multiforme
- Lupus erythematosus

➤ *Oral lichen planus* is the most frequent mucocutaneous disorder affecting the gingiva. Lesions may occur in isolation in the oral cavity or in combination with cutaneous lichen:
- Prevalence is between 0.1% and 4%; 65% of cases occur in women.
- In contrast to cutaneous lichen, oral lesions have a decidedly chronic course.
- Malignant transformation occurs in 0.5–2.5% of cases.
- Bilateral involvement of cheek mucosa and/or lateral sides of the tongue is usual.
- Appearance is polymorphic:
  - *Reticular and/or papular lesions* are very characteristic.
  - *Erosive lesions* emerge after rupture of blisters
  - *Atrophic* lesions develop especially following treatment with glucocorticosteroids.
  - *Plaque-like lesions* cannot be clinically differentiated from leukoplakia.
- Histopathology shows:
  - Subepithelial, band-like, mononuclear infiltrate mainly consisting of T lymphocytes and macrophages, characteristics of a type IV (delayed hypersensitivity) reaction
  - Degeneration of basal epithelial cells
  - "Saw-tooth" rete ridges of epithelium
  - Hyperortho- or hyperparakeratinization of epithelium
- Pathogenesis is still not well understood. It is presumed to be
  - a T-cell-mediated immunologic reaction to presently unknown antigen at the border between epithelium and connective tissue, or
  - a reaction to antigenically altered basal cells.
- Heterogeneous pathogenesis is possible:
  - So-called *lichenoid* reaction to:
    - dental materials such as amalgam, gold, composite resin
    - drugs, like gold salts, allopurinol, penicillamine, β-blockers, antimicrobial and antimalarial drugs, antihypertensives, sulfonylurea, etc.
- In association with graft-versus-host disease (GVHD) and hepatitis C

➤ *Pemphigoid:* a group of autoimmune diseases. Autoantibodies against components of the basal membrane are formed, leading to a split between epithelium and connective tissue:
- Bullous pemphigoid—predominately skin lesions; (oral) mucosal lesions are possible.

- Cicatricial pemphigoid—exclusively mucosal lesions, e.g., of the conjunctiva (with tendency to scar formation and possible blindness) or oral or genital mucosa:
  - Subepithelial bullae
  - IgG deposits and C3 located at the basal membrane
  - Serologically proven autoantibodies against keratinocytes may confirm the diagnosis.

➤ *Pemphigus vulgaris:*
- Autoimmune disorder with formation of autoantibodies against interepithelial adhesion molecules, resulting in destruction of desmosomes.
- Histopathology:
  - Acantholysis, intraepithelial vesicles
  - Free epithelial cells within the vesicle, so-called Tzank cells
  - Predominately mononuclear infiltrate with scattered neutrophilic granulocytes
  - Intercellular IgG autoantibodies
- Poor general prognosis with fairly high mortality

➤ *Erythema multiforme:*
- Acute vesiculobullous disease of, in particular, young individuals in their teens and 20s. Males are more frequently affected.
- There is a minor form with distinct, predominately skin lesions, and a major form (Stevens–Johnson syndrome) with widespread skin and mucous membrane lesions.
- Skin lesions present as erythematous papules, which enlarge and form central vesicles or bullae and a characteristic "target" or "iris" lesion.
- Possibly allergic immune-complex reaction to various factors:
  - Herpesviruses, *Mycoplasma pneumoniae*, streptococcal infection of the upper respiratory tract
  - Drugs: sulfonamides, phenytoin, pyrazolones, barbiturates, phenyl butazone, penicillin, carbamazepine.
- Self-limiting, infrequently recurrent disease.

➤ *Lupus erythematosus.* Autoimmune disorder of connective tissue. Formation of autoantibodies against various cell components (nucleus, cell membrane, etc.):
- Discoid lupus erythematosus—chronic form
- Systemic lupus erythematosus—severe form with organ involvement
- In both forms oral mucosa may be affected:
  - In cases of discoid lupus lesions are similar to leukoplakia or lichen planus.
  - Mucosal ulcerations occur in systemic lupus.

Allergic Reactions

➤ Allergic reactions which manifest themselves in the oral mucosa are very rare:
- Type I (anaphylactic) reaction:
  - Release of IgE by mast cells in response to ingredients in toothpaste, mouthwash, chewing gum
  - Acute inflammation of gingiva, ulceration
- Type IV reaction (delayed hypersensitivity, T-cell-mediated)
  - Contact allergy to dental materials such as mercury, nickel, gold, chromium, palladium, resin.
  - May manifest itself as lichenoid lesion. *Note:* Removal of material usually leads to resolution.

Traumatic Lesions

➤ Important elements in the differential diagnosis:
- General: self-inflicted (factitious), iatrogenic, or accidental
- Mechanical injury of gingiva by oral health remedies, dental instruments, or restorations
- Chemical injury: local application of certain chemicals such as aspirin, cocaine, pyrophosphates, detergents, chewing tobacco, bleaching agents
- Thermal injury, e.g. by hot food (pizza, coffee)

Foreign Body Reactions

➤ Acute or chronic inflammation of gingiva due to incorporated foreign material:
- Usually acute inflammation, gingival abscess
- Chronic foreign body reaction, e.g., amalgam tattoo

Periodontitis

Chronic Periodontitis

- Most frequent form of periodontitis. Infectious, inflammatory disease of the tooth-supporting apparatus with progressive attachment loss and loss of alveolar bone; cardinal symptoms are pocket formation and/or recession and gingival inflammation:
  - Occurs frequently after the age of 30 years; may also occur in children and adolescents.
  - Periodontal destruction correlates to the amount of local etiologic factors:
    - Frequently subgingival calculus.
    - Various associated microflora.
  - Slow or moderate progression. Periods of rapid progression may occur.
  - Further classification is based on the extent and severity of the disease:
    - *Localized* chronic periodontitis: ≤30% sites are affected.
    - *Generalized* chronic periodontitis: >30% sites are affected.
    - The severity of disease may be described for a single site, a single tooth, or the whole dentition: "slight": 1–2 mm attachment loss; "moderate": 3–4 mm; "severe": ≥5 mm attachment loss.
  - Not all cases respond to therapy: this is referred to as *refractory periodontitis*. The term does not designate a special disease category of chronic periodontitis.

Aggressive Periodontitis

- Infectious, inflammatory disease of the tooth-supporting apparatus with rapid attachment loss and loss of alveolar bone in otherwise healthy patients. Familial aggregation.
- Secondary features which are generally, but not universally, present:
  - Amount of microbial deposits is inconsistent with the severity of periodontal destruction
  - Elevated numbers and proportions of *A. actinomycetemcomitans* (and/or *P. gingivalis* in some populations) in subgingival plaque
  - Phagocyte abnormalities
  - Macrophage hyperresponsiveness, elevated levels of PGE_2 and IL-1β
  - Self-limiting in some cases
- Diagnosis is based on the history as well clinical and radiologic data. Laboratory testing may be helpful but is not essential for diagnosis.
- Aggressive periodontitis may be localized or generalized:
  - *Localized* aggressive periodontitis:
    - Onset around puberty
    - Robust serum antibody response to infecting agents
    - First molars/incisors are affected. Interproximal attachment loss on at least two permanent teeth, one of which is a first molar, and involving no more than two teeth other than first molars and incisors
  - *Generalized* aggressive periodontitis:
    - Onset usually before the age of 30 years but may be later
    - Poor serum antibody response to infecting agents
    - Episodic destruction of attachment and alveolar bone
    - Generalized interproximal attachment loss affecting at least three permanent teeth other than first molars and incisors
  - Additional descriptors may be risk factors, e. g., smoking, emotional stress, drugs, sex steroid hormones, etc.
  - Localized periodontitis occurring before onset of puberty may be classified either as localized chronic or localized aggressive periodontitis. Generalized forms of prepubertal periodontitis are virtually always a manifestation of systemic disease.

Periodontitis as Manifestation of Systemic Disease

- *Hematological disorders:*
  - *Acquired neutropenia, agranulocytosis:* mucosal necrosis and severe periodontal disease may occur during periods of markedly decreased numbers of neutrophilic granulocytes.
  - *Leukemia:* periodontal lesions, particularly in acute forms of leukemia:
    - Gingival enlargement due to leukemic cell infiltrates, particularly acute monocytic leukemia, or chronic lymphatic leukemia
    - Gingival necrosis

- *Genetic diseases:*
  - *Familial benign, cyclic, and chronic neutropenia:* more protracted forms with advanced periodontitis and generalized bone loss. Periodontal destruction may be limited by good oral hygiene and intensive dental care.
  - *Down syndrome (trisomy 21):* generalized periodontitis which starts developing before the age of 10 years and is characterized by rapid progression. Complete loss of teeth by the age of 30 years is possible.
  - *Leukocyte Adhesion Deficiency Syndromes:* rare, autosomal recessive diseases with poor overall prognosis. Expression of adhesion molecules CD11a/CD18, CD11b/CD18 and CD11c/CD18 suppressed (cf. Fig. 3.**4**). Generalized periodontitis in the first dentition; considerable gingival inflammation with gingival proliferation and development of clefts.
  - *Papillon-Lefévre syndrome:* rare (about one case in one to four million), autosomal recessive inheritance characterized by hyperkeratotic skin lesions (palmoplantar hyperkeratosis) associated with severe generalized periodontitis. Several mutations in the cathepsin C gene located in chromosome 11q14–q21 may lead to complete loss of enzymatic activity; respective mutations may also be present in cases of prepubertal periodontitis independent of the syndrome. Periodontitis affects both dentitions.
  - *Chediak–Higashi syndrome:* very rare autosomal recessive disease with poor overall prognosis. Functional impairment of neutrophilic granulocytes (chemotaxis, intracellular killing).
  - *Glycogen storage disease:* autosomal recessive condition. Faulty carbohydrate metabolism associated with neutropenia and impaired function of neutrophilic granulocytes.
  - *Infantile genetic agranulocytosis:* very rare autosomal recessive disorder with severe neutropenia. Generalized periodontitis of the first dentition.
  - *Cohen syndrome:* autosomal recessive disease characterized by nonprogressive mental and motor retardation, obesity, dysmorphia, and neutropenia. Affected individuals have more periodontitis than matched controls.
  - *Ehlers–Danlos syndrome:* autosomal dominantly inherited group (10 types) of diseases characterized by impaired collagen synthesis affecting mainly the skin and joints. Types IV and VIII are associated with severe periodontitis of the first dentition.
  - *Hyperphosphatasia:* autosomal recessive deficiency of alkaline phosphatase. Severe periodontitis of the first dentition.
- *Histiocytosis syndromes:*
  - *Abt–Letterer–Siwe syndrome:* sepsis-like disseminating (neoplastic) histiocytosis in infants and small children with skin and organ involvement. Fatal.
  - *Hand–Schüller–Christian disease:* chronic disseminating histiocytosis in children and adolescents. Bone, skin, and organ lesions. Poor overall prognosis.
  - *Langerhans cell tumor* (eosinophilic granuloma):
    - Solitary or multiple bony lesions without skin or organ involvement
    - Mildest form of the disease, which may imitate localized aggressive periodontitis or necrotizing periodontal diseases
    - Biopsy necessary to confirm suspected diagnosis. Thorough general examination mandatory
    - Therapy: extraction of extremely mobile (floating) teeth, curettage of bony lesions, in some cases low-dose radiation

Necrotizing Periodontal Diseases

General Remarks

- Various forms are distinguished depending on the extent of the disease and the topography of the lesions :
  - *Necrotizing ulcerative gingivitis:*
    - Epidemic-like dissemination in World War I in soldiers fighting in the trenches ("trench mouth")
  - *Necrotizing ulcerative periodontitis:*
    - Rapid attachment loss, usually without pocket formation
    - Bony sequestration may occur
    - Close association with HIV infection

- *Necrotizing stomatitis:*
  - Occurring independently or secondary to necrotizing ulcerative gingivitis/periodontitis
  - In populations with very poor food supply and hygiene, noma (cancrum oris) may develop, characterized by severe soft and hard tissue destruction and high risk of facial mutilation.

Necrotizing Ulcerative Gingivitis

➤ Acute, painful disease confined to gingiva. Symptoms are:
- abrupt onset of *pain*;
- *necrosis* of the tip of the interdental papilla;
- *ulcer* formation with white-yellowish or gray pseudomembranes, demarcated by linear erythema;
- *halitosis*;
- regional lymphadenitis and subfebrile condition may occur;
- peak incidence in patients in their 20s.

➤ Etiology:
- Invasive gram-negative, obligate anaerobes:
  - Spirochetes
  - Fusobacteria
  - *P. intermedia*
- Presumed association with herpesviruses, e.g., human cytomegalovirus

➤ Risk factors:
- Poor oral hygiene
- Cigarette smoking
- Psychological stress, lack of sleep
- HIV infection

➤ Histopathology:
- Nonspecific, acute necrotizing inflammation.
- Ulcerated areas are covered by pseudomembranes consisting of a network of fibrin, perished epithelial cells, leukocytes, erythrocytes, and bacteria.
- Connective tissue beneath contains widened and proliferating vessels and a dense infiltrate of neutrophilic granulocytes, plasma cells, and macrophages.
- Invasive fusobacteria and spirochetes.

Necrotizing Ulcerative Periodontitis

➤ Acute periodontal infection characterized by an expansion of necrotic lesions into the periodontal ligament and alveolar bone. Usually occurs as a manifestation of severe weakness of the immune system (immune suppression, nutritional deficit):
- Rapid attachment loss, often without formation of deep pockets, but with the possibility of bony sequestration
- Closely associated with HIV infection/AIDS:
  - *Note:* Necrotizing ulcerative periodontitis in HIV-seropositive patients is a sign of <200/µm $CD4^+$ cells
  - Prognostic indication of the development of AIDS

Abscesses of the Periodontium

Gingival Abscess

➤ Definition: acute inflammatory condition of the gingiva characterized by purulent exudate without attachment loss:
- Following traumatic insult, e.g., injury by a fish bone, interdental brush, etc.:
  - implantation of virulent bacteria into gingival connective tissue leads to
  - excessive inflammatory reaction.
- In the context of hormonally exacerbated gingivitis:
  - Pregnancy gingivitis, or gingivitis associated with contraceptives
  - Gingivitis associated with diabetes mellitus
- In the context of drug-induced gingival enlargement

Periodontal Abscess

➤ Acute exacerbation of already existing marginal periodontitis:
- Increase in numbers of virulent bacteria such as *P. gingivalis*, *P. intermedia*, *F. nucleatum*, *T. forsythia* in the subgingival environment; penetration of the pocket wall
- Local and general immune deficiency
- Obstruction of the pocket entrance due to complicated morphology, e.g., furcation or intrabony pocket
- Frequently seen in the final stage of periodontal disease

- Occasionally *multiple periodontal abscesses* develop:
  - Exacerbation of generalized periodontitis, e. g., following administration of oral penicillins which are not β-lactamase-resistant.
  - In patients with diabetes mellitus
  - In individuals with impaired immune systems.
  - *Note:* Differential blood count must be ordered in any case of multiple periodontal abscesses.

Pericoronal Abscess

- Localized purulent infection within the tissues surrounding the crown of a partially erupted tooth. Mandibular wisdom teeth are particularly affected. Peak incidence between 18 and 24 years of age.
- Symptoms are:
  - Red and swollen gingiva, purulent exudate
  - Mouth opening is painful and frequently limited
  - Lymphadenitis
  - Inflammation may spread into the neighboring spaces:
    - posteriorly into the oropharyngeal space
    - medially to the base of the tongue (sublingual abscess, peritonsillar abscess, perimandibular abscess)
  - *Note:* Commensal periodontal pathogens may multiply excessively in the area of erupting wisdom teeth even without symptoms.

Combined Periodontic-Endodontic Lesions

- Close periodontic-endodontic interrelationships exist:
  - via the apical foramen;
  - via lateral and furcal foramina;
  - via dentinal tubuli after removal of root cementum.
- Infections may spread from one area into the other:
  - Retrograde pulpitis, if and when the plaque front within a periodontal pocket reaches the apex or accessory pulp channels
  - Spread of an endodontic (periapical) infection into the periodontal ligament

Developmental or Acquired Deformities and Conditions

Localized, Tooth-Related Factors That Modify or Promote Plaque-Induced Gingival Diseases/Periodontitis

- Anomalous position of teeth in the dental arch and tooth anatomy, dental restorations and appliances, root fractures, cervical root resorption, and cement tear may increase the risk of developing local periodontal disease. In the last cases in particular, explorative surgery may be necessary for diagnosis.

Mucogingival Deformities and Conditions Around Teeth and on Edentulous Alveolar Ridges

- Mucogingival deformities may occur around teeth and in edentulous areas of the alveolar ridge. In addition to the classification outlined in Table 6.**1**, severity and etiological characteristics may be used to describe the lesions and conditions. Recessions in particular may be localized or generalized.

Occlusal Trauma

- Excessive occlusal forces do not lead to plaque-associated periodontal diseases and attachment loss, but they may accelerate the progression of already existing periodontitis:
  - *Primary occlusal trauma:* periodontal injury due to excessive occlusal forces in a normal tooth-supporting apparatus (normal bone and attachment level)
  - *Secondary occlusal trauma:* periodontal injury due to normal or excessive forces in a reduced periodontium (loss of bone and attachment)
- *Note:* Increased tooth mobility (which is not synonymous with occlusal trauma) can have a negative impact on periodontal conditions and, especially, treatment results.

Diagnosis

7 Diagnosis of Periodontal Disease

History

General Remarks

- At the heart of current dental treatment philosophy is comprehensive dental care. Careful examination of a patient usually yields numerous different diseases and conditions in the oral cavity, for instance:
  - Caries and endodontic lesions
  - Periodontitis and other chronic infections
  - Orthodontic and orthognathic disorders
  - Temporo-mandibular disorders
  - Diseases of the oral mucosa
- Within the context of comprehensive therapy, the dentist should generally strive for both esthetic and functional rehabilitation. The aim is to reach an optimum situation under conditions of health which *meets the patient's needs* with regard to esthetics and chewing comfort.
- An important aspect is planning treatment that is *appropriate* to each individual case, avoiding both under- and overtreatment.
- The main points in treating periodontally diseased patients are:
  - anti-infection therapy;
  - regenerative measures;
  - risk factor modification.
- Before any therapy begins, detailed *diagnosis* is mandatory, which must be based on specific information of the patient's *history* and comprehensive clinical and, if necessary, radiological and/or laboratory *examination*. Relevant findings must be recorded appropriately. *Note:* Appropriate diagnostic measures must be carried out during all treatment phases.

Medical History

- Information on the patient's *general history* can be gathered in the waiting room by means of a questionnaire which the patient fills out. The patient's answers are checked and discussed in more detail during the *dental consultation*.
- The history is obtained in a systematic manner (Fig. 7.**1**). From the first visit, existing and potential risk factors for destructive periodontal diseases and conditions which might interfere with the treatment itself, patient management, or the expected therapeutic result should be taken account of, especially the following:
  - Infectious diseases
  - Increased risk of endocarditis
  - Allergies and incompatibilities
  - Cardiovascular diseases, high blood pressure
  - Diabetes mellitus
  - Pregnancy
  - Osteopenia, osteoporosis
  - Drugs, drug abuse
  - Smoking behavior
  - Stress and coping behavior
- During the consultation, further important information will be acquired—often almost unconsciously—about the physical and psychological condition of the patient.
- If necessary, the patient should be referred to a specialist. *Note:* Periodontally diseased patients present more frequently with systemic diseases, which can interfere with treatment planning.

Dental History

- The first question is about the reason for consultation:
  - Pain or alarming symptoms, such as tooth mobility, tooth migration, bad breath, bad taste
  - Esthetic problems
  - Desire for comprehensive treatment
- Next, questions are asked about the following:
  - Accidents, injuries, surgical operations on the head and neck
  - Previous orthodontic treatment

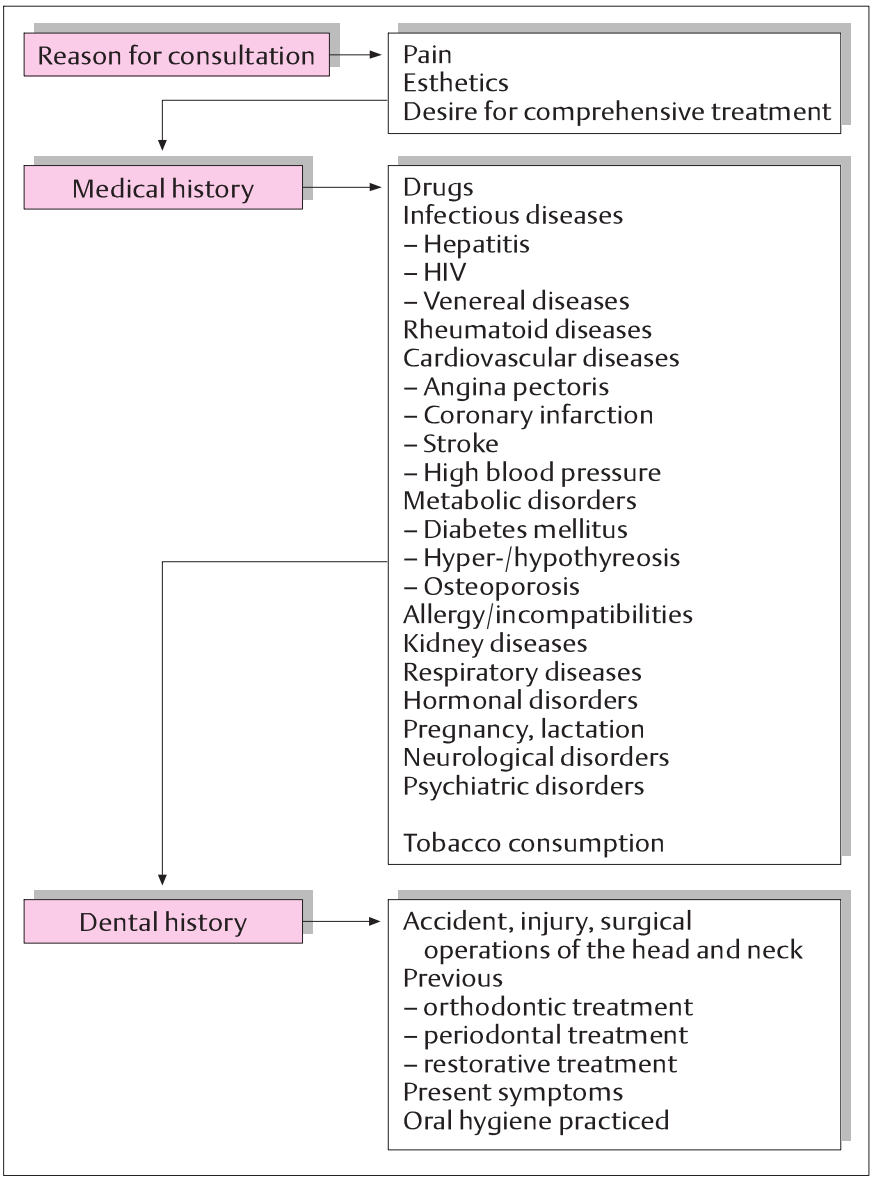


Fig. 7.1 Systematic medical and dental history

- Previous periodontal treatment
- Dental restorations
- Practice of oral hygiene

Clinical Examination

Extraoral Examination

➤ The clinical examination generally starts extraorally:
- Color and perfusion of the skin, assessment of the lip mucosa
- Asymmetries in the head and neck region
- Palpation of submandibular and sublingual lymph nodes
- Palpation of foramina of the trigeminal nerve
- Eyes: conjunctiva, nystagmus, exophthalmos
- Hands: tremor, clubbing of the last digits of fingers, sweat

Intraoral Examination

➤ First, the mucous membranes are inspected. Using a dental mirror, the condition of masticatory, lining, and specialized mucosa are assessed, beginning in the pharyngeal region. A systematic *stomatological examination* is especially important for cancer screening

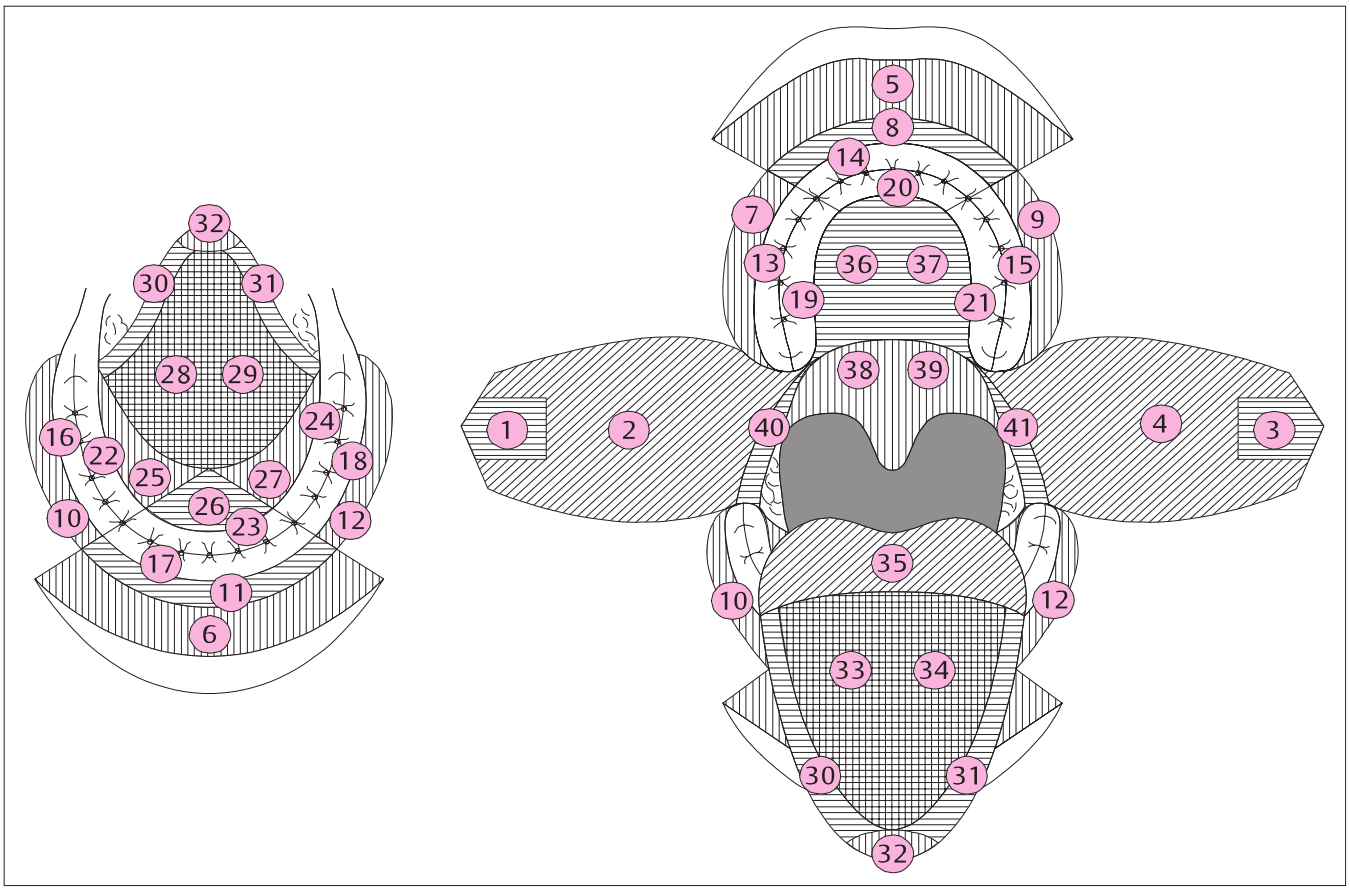


Fig. 7.**2** Form for the documentation of oral mucosal lesions (Roed-Pedersen and Renstrup 1969). Teeth that are present are marked by circles (mandible *left*, maxilla *right*). White alterations are marked in *black: 1* striae; *2* plaque-like lesions; *3* papular lesions. Red alterations are marked in *red: 1* slight erythema; *2* overt erythema; *3* bulla; *4* desquamation/ulceration

and should be documented on a special form (Fig. 7.**2**):
- Lips
- Tonsils, pharynx
- Mucosa of the soft and hard palate
- Cheek mucosa
- Dorsum and lateral sides of the tongue, floor of the mouth
- Gingiva: form, color, consistency
- Flow rate and consistency of saliva

➤ *Dental examination:*
- Missing teeth
- Restored tooth surfaces (marked in blue)
- Carious tooth surfaces:
  - Invasive therapy necessary (marked in red)
  - Lesions to be monitored for caries after preventive measures are started (may be marked in green)
- Other defects of the solid substance of the tooth: erosions, attrition, abrasion
- Results of sensitivity testing, e.g., with carbon dioxide ice
- Dentin hypersensitivity
- Tenderness to percussion

➤ Photographic documentation:
- For forensic reasons
- To document the condition before, during and after therapy:
  - Focal length between 90 and 120 mm
  - Ring flash
  - Anterior teeth: jaws closed, ×2/3
  - Left and right lateral aspects: jaws closed, ×2/3
  - Occlusal aspects of the upper and lower jaws with a mirror: ×1/2
  - If necessary, details at higher magnification
- Polaroids of intraoral pictures may also be useful for patient motivation.

Functional Examination

- *Clinical function analysis* during basic examination:
  - Palpation of temporomandibular joints
  - Joint sounds (clicks, crepitation)
  - Palpation of jaw and face muscles
  - Distance between incisal edges at maximum mouth opening
  - Deviation of the mandible during mouth opening
  - Occlusal interferences during
    - retrusion of the mandible;
    - protrusion;
    - laterotrusion: working side contacts, balancing contacts.
  - Wear facets, signs of bruxism
- Creation of stone model casts for planning:
  - Alginate impressions with rimlock trays:
    - Impression of the vestibule including lip and cheek frenula
    - Complete representation of the occlusal conditions
  - Maxillomandibular registration record in maximum intercuspidation (e.g., wax registration)
  - Stone model casts, if necessary mounted in a semiadjustable articulator

Periodontal Examination

- Table 7.**1** lists the standard instruments for the periodontal examination tray.
- Periodontal screening and recording by sextant (PSR, see Table 4.**4**) may be performed:
  - PSR should ensure that periodontal lesions are not overlooked.
  - However, it cannot replace the detailed periodontal examination necessary in patients with periodontally disease.
- Cardinal symptoms of inflammation:
  - *Redness* of the gingival margin
  - *Edematous swelling*
  - Compared to the sublingual area, *temperature* may be increased, and can be measured with appropriate probes (PerioTemp)
  - *Pain*, especially in cases of ulceration and/or abscess formation
  - *Impaired function*, e.g. increasing tooth mobility and tooth migration.
- Important additional findings:
  - Tendency to *bleed* on being probed
  - Inflammatory, sometimes purulent, gingival exudate
- Periodontal findings are recorded on four or six—sometimes even more—surfaces of all teeth, e.g.:
  - Mesiobuccally
  - Midbuccally
  - Distobuccally
  - Distolingually/distopalatally
  - Midlingually/midpalatally
  - Mesiolingually/mesiopalatally
- The following are assessed with *periodontal probes:*
  - Ecological niches for periodontal pathogens
  - Local destruction of the tooth-supporting apparatus
  - Bleeding tendency of the gingiva

Table 7.**1** Standard instruments for periodontal examination tray

Instruments	Description	Article no., manufacturer
Mouth mirror	Plane, front surface rhodium-coated, ∅ 22 mm Mirror handle	M4C, Hu-Friedy MHE6, Hu-Friedy
Dressing pliers		DP18 or DP17, Hu-Friedy
Explorer	• Double-ended • For subgingival calculus detection • For caries detection	EXD5, Hu-Friedy
WHO probe	• Ball-shaped tip • WHO markings (3.5 mm, 5.5 mm)	PCP11.5B, Hu-Friedy
Periodontal probe	Calibration in 1 mm steps, or color coded 3–3–2–3 mm	PCPUNC15 or PCP11, Hu- Friedy
Furcation probe	Curved Nabers probe, calibration in 3–3–3–3 mm steps	PQ2N, Hu-Friedy

- Various probes may be employed:
  - Rigid, color-coded, not pressure-controlled periodontal probes, e.g.:
    - ø 0.4–0.5 mm, e.g., PCP 11, PUNC 15 (Hu-Friedy, Leimen, Germany)
    - Recommended probing force 0.25 N; the pressure may be estimated as about 2 MPa ($P = F/r^2\pi = 0.25/0.2^2\pi$ N/mm^2).
  - Simple pressure-calibrated probes, e.g., ClickProbe (KerrHawe, Bioggio, Switzerland) are particularly used for a more objective assessment of gingival bleeding tendency.
  - Computer-assisted automatic probes, e.g., Florida probe, PeriProbe (Vivacare, Schaan, Liechtenstein), are only used for research.
- In untreated patients, probing depth should never be assessed with pressure-controlled probes:
  - Subgingival calculus may obstruct the entrance to the pocket.
  - Therefore, forced probing is better.
- *Note:* Periodontal probing is unpleasant or even painful, particularly if the gingiva is inflamed. The patient needs to be prepared for this.
- *Definitions:*
  - *Periodontal probing depth:* distance between the gingival margin and the clinically probeable bottom of the pocket or sulcus
  - *Clinical attachment level:* distance between the cementoenamel junction and the clinically probeable bottom of the pocket or sulcus (Fig. 7.**3**). *Note:* The real bottom of the lesion or the sulcus bottom cannot be determined with periodontal probes:
    - When the gingiva is inflamed the probe will always pass the junctional epithelium; at 2 MPa it is already in the connective tissue.
    - On the other hand, the probe cannot pass a long junctional epithelium, which might have formed after periodontal treatment.
  - *Histological attachment level:* distance between the cementoenamel junction and first fibers of the supraalveolar fiber apparatus.
  - *Gingival recession:*
    - Difference between clinical attachment level and periodontal probing depth.
    - If there is a negative difference, recession is 0.
- *Furcation diagnosis.* Periodontal lesions which reach the furcation area have a marked horizontal component. The degree of severity is of prognostic importance.
  - Probing should be with a curved, color-coded probe, e.g., Nabers probe PQ2N (Hu-Friedy, Leimen, Germany) with calibration in 3-mm steps.
  - Measurements are done from an imaginary tangent connecting the prominences of the root surfaces of both roots (Fig. 7.**4**):

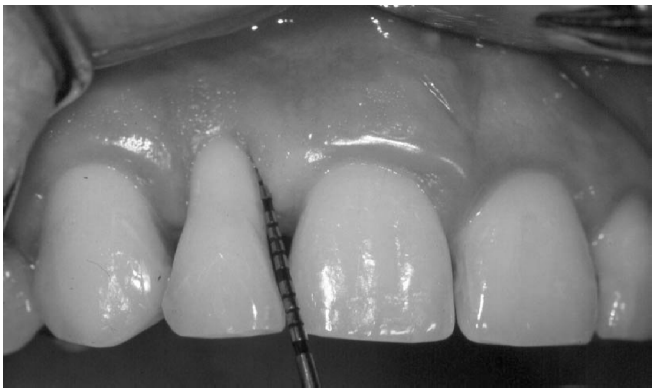


Fig. 7.**3** Periodontal probing. The probe (PCPUNC15) is calibrated in one-millimeter steps. Periodontal probing depth amounts mesiobuccal to tooth 12 to about 3.5 mm. Clinical attachment level is 7.5 mm. That means that, at this site, recession amounts to 4 mm.

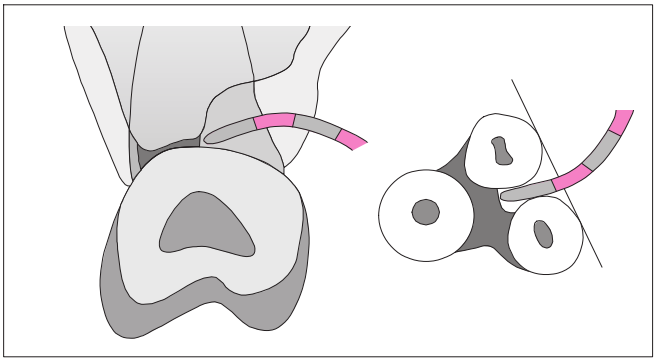


Fig. 7.**4** Probing of the furcation area. Measurements are performed with a curved probe (Nabers probe) to an imaginary tangent which connects the prominences of both roots. Since the furcation entrance is usually in a subgingival location, the severity of furcation involvement can only be estimated. A more detailed assessment is necessary during surgery

- Degree I: horizontal attachment loss up to 3 mm
- Degree II: horizontal attachment loss of more than 3 mm, but not encompassing the whole furcation area
- Degree III: through-and-through involvement

– Detailed knowledge of the location of furcation entrances is necessary. *Note:* The entrances need to be actively sought, so forced probing is recommended:
- In maxillary molars: buccally, mesiopalatally, and distopalatally
- In maxillary first premolars: mesially and distally, from both the buccal and the palatal aspect
- In mandibular molars: buccally and lingually

– The entrance of a furcation is usually located subgingivally. The involvement can therefore only be grossly estimated. More detailed assessment is performed intraoperatively.

➤ *Assessment of tooth mobility:*
– Manual estimation, e.g., using two instrument handles:
- Degree I: crown can be tilted up to 1 mm horizontally
- Degree II: crown can be tilted more than 1 mm horizontally
- Degree III: tooth moves horizontally and vertically in response to cheek or tongue pressure. Function is severely impaired.

– Electronic measurement (PerioTest). The tooth's dampening characteristics are assessed.
– *Note:* Tooth mobility is not in itself a prognostic factor.

➤ *Bleeding on probing* (Fig. 7.**5**):
– Greater density of vessels and loss of collagen within the infiltrated connective tissue of the gingiva may lead to bleeding after minimal mechanical injury.
– Various findings:
- Bleeding after probing the marginal gingiva (sulcus bleeding): evidence for the influence of supragingival plaque on marginal tissues. May be assessed with index systems (e.g., SBI, PBI, GBI) to document development and course of periodontal therapy (Fig. 7.**5 a**).
- Bleeding on probing to the (probeable) bottom of the pocket or sulcus. Indicates the presence of subgingival plaque. This finding should always be recorded in combination with the periodontal probing depth (Fig. 7.**5 b**).

➤ *Purulent exudate:* this sign of severe inflammation, from which in the last century the term "pyorrhea alveolaris" was derived, is rarely observed nowadays.

➤ All periodontal findings must be documented in a suitable chart (Fig. 7.**6**).

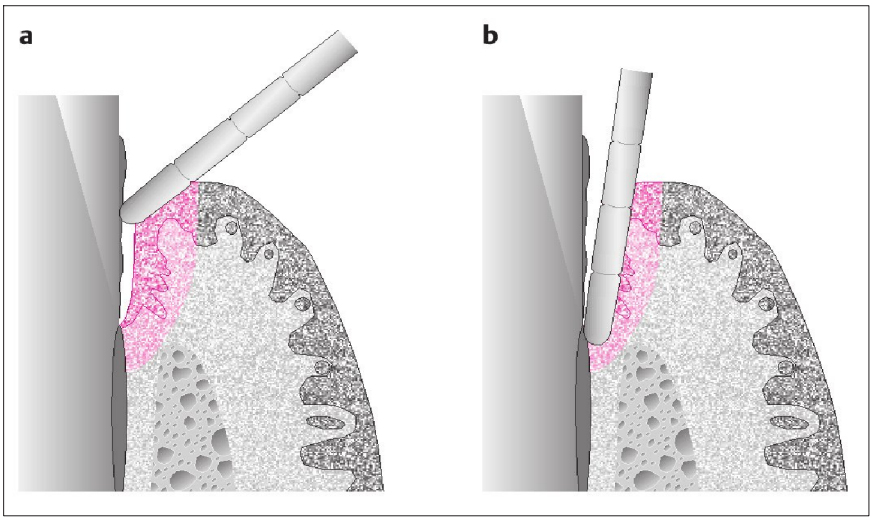


Fig. 7.**5** Bleeding on probing.
a Bleeding tendency of the gingival margin (e.g., PBI, SBI, GBI). The probe is carefully inserted into the sulcus and run around the tooth at an angle of about 45°.
b Bleeding on probing to the (probable) bottom of the pocket or sulcus

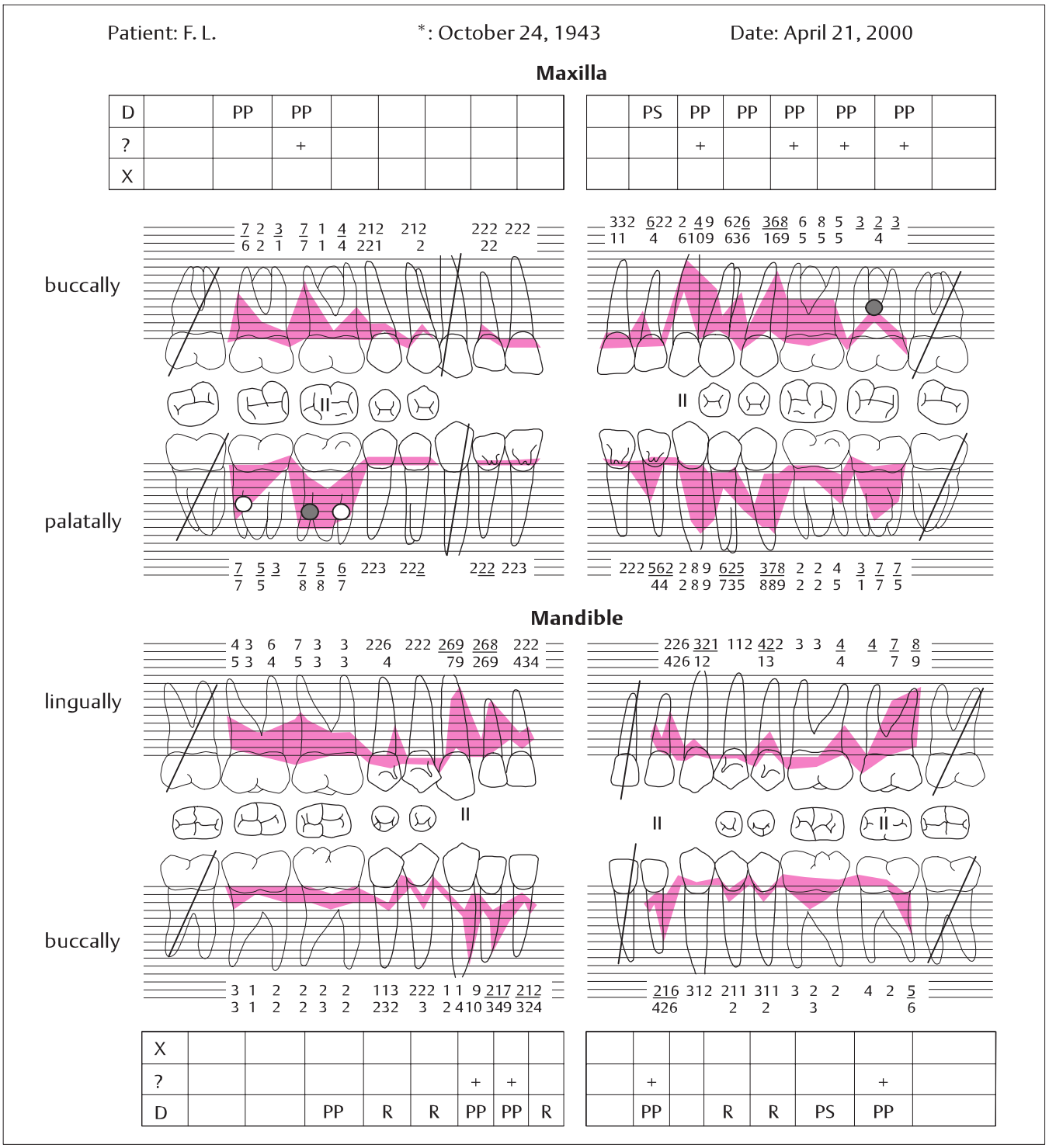


Fig. 7.**6** Filled-in periodontal chart. Missing teeth, periodontal probing depths (*upper row* on each side), clinical attachment levels (*lower row* on each side; only attachment loss is recorded), bleeding on probing (underlined probing depth) and gingival margin (recession). Additional recordings (if applicable): caries and restorations, sensitivity, tooth mobility (*roman numerals*), furcation involvement: incipient furcation involvement (degree I), ● advanced furcation involvement (degree II or through-and-through). Diagnosis (D): G: gingivitis, R: recession, PS: superficial periodontitis, PP: profound periodontitis. Preliminary prognosis (X: considered for extraction, ?: questionable prognosis)

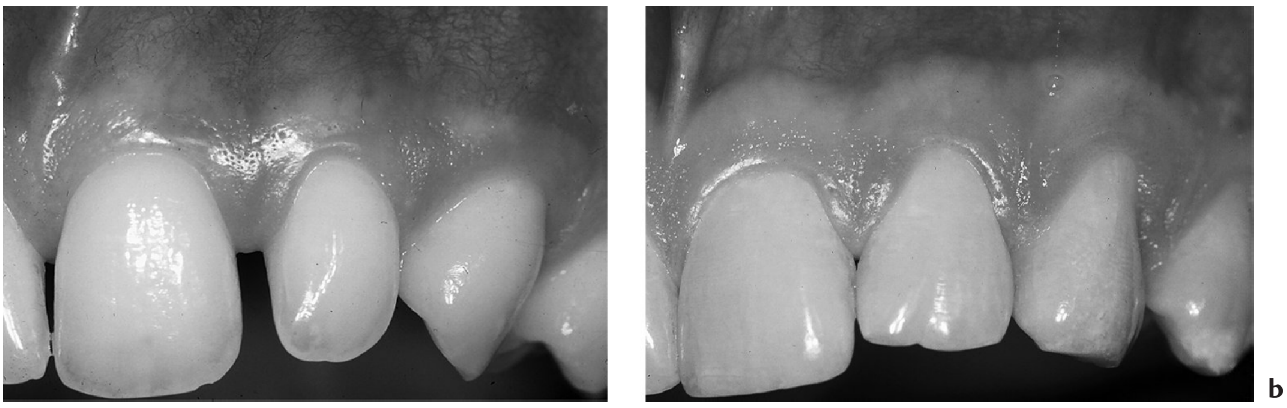


Fig. 7.**7** **a** "Thin" periodontal phenotype: slender anterior teeth in the maxilla with a narrow band of gingiva which appears thin and fragile. **b** "Thick" periodontal phenotype: rather wide, square anterior teeth. Gingiva is considerably wider and appears thicker

Mucogingival Examination

➤ In patients presenting with periodontal esthetic deficits and/or gingival recession, a detailed examination of the mucogingival situation is necessary, taking account of:
- The depth of the vestibule
- Aberrant lip and cheek frenal insertions
- The *position* of the gingiva in relation to the cementoenamel junction
- The *width* of the gingiva:
  - Contrast may be enhanced by staining the glycogen-rich alveolar lining mucosa with an aqueous solution of 1 % iodine/2 % potassium iodide.

Fig. 7.**8** Classification of gingival recessions according to Miller (1985). Class I (**a**) and class II (**b**) recessions may be covered up to 100 %, unlike class III (**c**) and class IV (**d**) recessions, where respectively partial or no root coverage is possible

- The distance between the gingival margin and the mucogingival border may be measured with a periodontal probe or caliper.
- Subtraction of the probing depth yields the width of the *attached gingiva.*

– The *thickness* of gingiva may be measured by ultrasound (Krupp SDM, Austenal, Cologne, Germany)
– Excessive gingival display, length of the upper lip
– Assessment of the *periodontal phenotype:*
  - Narrow band of rather thin gingiva (<1 mm), scalloped contour of the alveolar bone. Fragile gingiva with a tendency to recession. Frequently associated with slender anterior teeth in the maxilla (Fig. 7.**7 a**).
  - Thick (about 1.5 mm) and wide gingiva (3–5 mm), wide interdental septa. Thickness and width is usually combined with rather square anterior teeth in the maxilla. This phenotype has a low tendency to recession development (Fig. 7.**7 b**).

➤ Gingival recession
– Depth and width of recession as measured with a periodontal probe or caliper
– Loss of papilla
– Classification according to Miller (1985) (Fig. 7.**8**):
  - Class I: marginal tissue recession not extending to the mucogingival border; no loss of interdental bone or soft tissue. Complete coverage may be obtained with certain surgical methods.
  - Class II: marginal tissue recession extends to or beyond the mucogingival border; no loss of interdental bone or soft tissue. Complete coverage may be obtained with certain surgical methods.
  - Class III: marginal tissue recession extends to or beyond the mucogingival border; loss of interdental bone, or soft tissue is apical to the cementoenamel junction, but coronal to the apical extent of the marginal tissue recession. Partial coverage may be obtained.
  - Class IV: marginal tissue recession extends beyond the mucogingival border; loss of interdental bone extends to a level apical to the extent of the marginal tissue recession. Cannot at present be covered surgically.

– Stillman clefts (Fig. 7.**9 a**):
  - Very narrow, comma-shaped recession within the marginal gingiva
  - Caused by trauma, usually during horizontal tooth brushing

– Gingiva may be thickened around recession sites: fibrous enlargement as a response to frequent toothbrush injury (Fig. 7.**9 b**).

➤ All findings are documented in a special recession chart (Fig. 7.**10**).
➤ Alveolar ridge defects may occur in edentulous parts of the jaw:
– Class I: *buccolingual* loss of bone, alveolar process is of normal height
– Class II: *loss of height* of the alveolar process, normal buccolingual width

a b

Fig. 7.**9**
a Class I recession at canine with a Stillman cleft.
b Class I recession at canine with a fibrous enlargement of the gingiva

Patient: B. G. *: 2/14/1975 Date: 6/17/2000

16	15	14	13	12	11		21	22	23	24	25	26
			4.5	2	0	Recession depth	2.5	0	3.5			
			4	2	0	Recession width	3.5	0	4.5			
			1	1	2	Probing depth	2	1	2			
			0	2	3.5	Gingival width	2	3	0			
			1.2	0.9	1.3	Gingival thickness	1.2	1.0	1.2			
			II	I	0	Miller class	III	0	IV			
		II				Miller class						
		0.8				Gingival thickness						
		0.5				Gingival width						
		I				Probing depth						
		3				Recession width						
		2.0				Recession depth						
46	45	44	43	42	41		31	32	33	34	35	36

Fig. 7.**10** Filled-in recession chart. Note FDI numbering of teeth.

– Class III: *combined loss* of width and height of the alveolar process

Oral Hygiene

➤ Calculus and the topographical distribution of supragingival plaque and inflamed gingival units are documented in special forms:
  – to motivate and inform the patient about progress;
  – to document any changes in oral hygiene level.

➤ After disclosure with, e.g., 3 % aqueous solution of erythrocin the presence of plaque on four surfaces of every tooth (mesial, buccal, distal, and lingual) is recorded using, e.g., the Plaque Control Record (PCR, O'Leary et al. 1972).

➤ Similarly, the presence of bleeding after careful probing of the sulcus is recorded (cf. Fig. 7.**5 a**), e.g., Gingival Bleeding Index (GBI, Ainamo and Bay 1975):
  • Starting, e.g., distobuccally, the probe is inserted slightly into the sulcus and run to the buccal and mesial surfaces of the tooth at an angle of about 45°. This is repeated for every tooth.
  • Probing is similarly carried out at palatal/lingual sites.
  • Any gingival units that bleed are recorded.

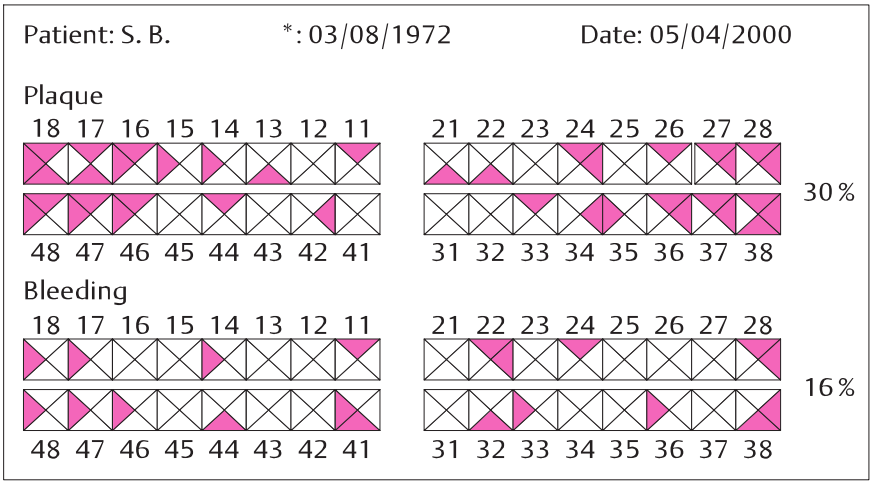


Fig. 7.**11** Topographical distribution of plaque-covered tooth surfaces (PCR = 30 %) and gingival units bleeding after marginal probing (GBI = 16 %). Note FDI numbering or teeth.

➤ The percentage of tooth surfaces covered by plaque and the percentage of gingival units that bleed after probing of the marginal gingiva are calculated. The patient is told the result, which is documented in the file (Fig. 7.**11**).

Radiological Examination

General Remarks

➤ To keep radiation exposure to a minimum, radiological examinations should only be carried out for an appropriate indication, e.g., to confirm or supplement uncertain clinical findings.

➤ *Note:* In cases of advanced periodontal disease, detailed radiological study is mandatory.

➤ The following parameters are assessed:
- Absolute bone loss—the distance (in millimeters) between the cementoenamel junction and the alveolar bone crest or bottom of the bony lesion. Note that, because of the supraalveolar fiber apparatus, values within 1.5–2 mm are normal and do not represent bone loss.
- Relative bone loss—in relation to the root length (percentage of bone loss).
- Bony pockets.
- Desmodontal space.
- Endodontic situation and periapical area.
- Number and shape of roots, topography of the root complex.
- Furcation involvement.
- Adjacent areas:
  - Maxillary sinus
  - Inferior alveolar nerve
  - Mental foramen
- Additional findings may include:
  - impacted teeth;
  - remaining roots;
  - cysts;
  - root resorptions;
  - hypercementosis.

➤ Radiographs taken at regular intervals allow comparisons between earlier and later exposures.

Panoramic Radiographs

➤ Orthopantomogram (OPT)—provides a general overview, which may be sufficient for periodontal diagnosis in mild or moderate cases.
- Advantages:
  - Low radiation dose.
  - Neighboring structures such as temporomandibular joints, maxillary and nasal sinus, and impacted teeth can be assessed.
- Disadvantages compared to intraoral radiographs:
  - Images not in the plane of interest are blurred and distorted.
  - Uneven enlargement.
  - In anterior regions the cervical spine is superimposed.
- *Note:* Periapical or periodontal lesions of the furcation area can sometimes be more easily detected on an OPT than on intraoral radiographs:
  - OPT is a tomogram which preferentially images the central plane of the alveolar process.
  - This is the layer that usually contains these pathological structures.

Full-Month Radiographic Survey

➤ A fullmonth radiographic survey is mandatory in patients with advanced periodontitis and in those undergoing comprehensive therapy:
- Standardized *paralleling technique* using special film holders with adjustment device and a long cone (Fig. 7.**12 a**). The central X-ray beam should be perpendicular to the film.
- Ultraspeed (D) films yield a more detailed image because the grain size is smaller.
- Ektaspeed (E) films need only half the exposure radiation because they have a larger grain size.
- *Note:* Studies of the ability to diagnose lesions on the basis of D or E films did not show any difference.
- Low kilovoltage (65–70 kV) is best to obtain more contrast, e.g., to locate the tip of an endodontic instrument in a root canal.
- High kilovoltage (90 kV) yields films with lower contrast, preserving more information regarding, e.g., bone density and the height of the bone crest.

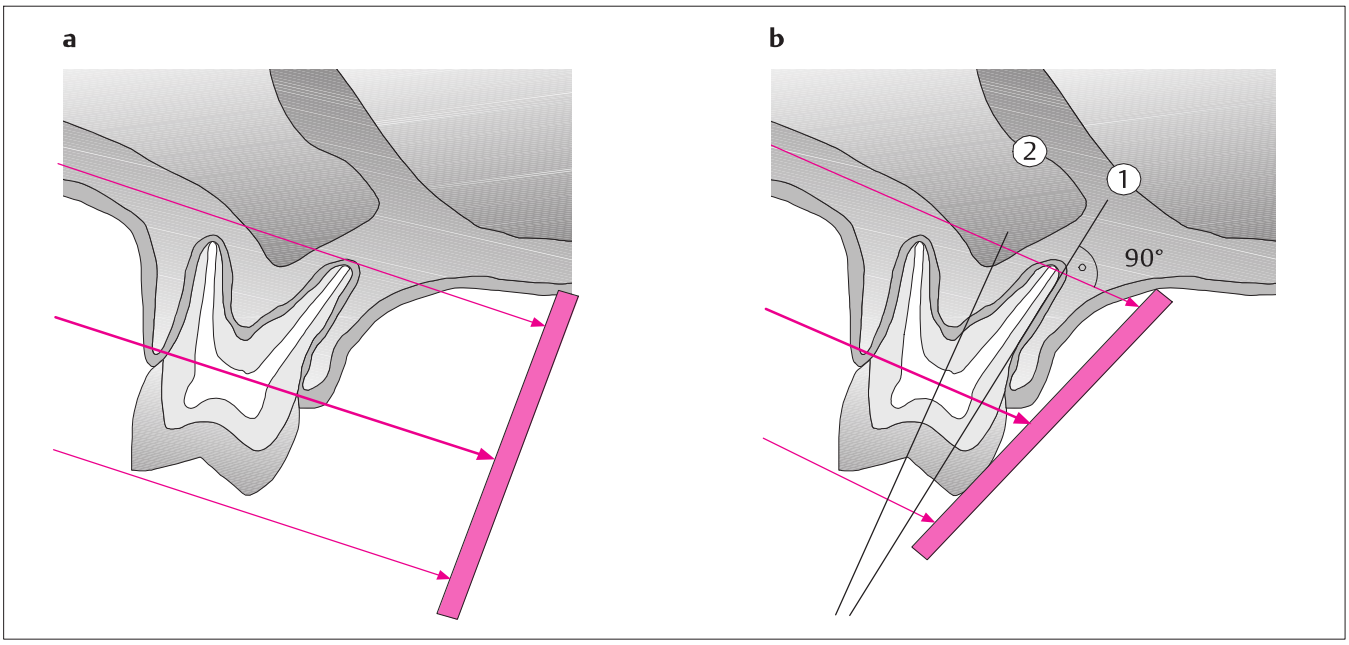


Fig. 7.**12**
a Paralleling technique. The film is positioned with a special film holder quite far from the tooth and parallel to its long axis. The long cone of the X-ray machine is adjusted perpendicular to the film. The tooth image corresponds to the actual size of the tooth.
b Bisecting-angle technique. Similar results can be obtained if the film is positioned close to the tooth and the long cone is positioned so that the central beam is perpendicular to the bisecting plane (1) of the angle between the film and the tooth axis (2) but this technique is not very reliable and is not recommended

➤ Periapical X-ray survey:
- Fourteen periapical exposures (Fig. 7.**13**):
  - Two exposures for each molars and premolars in the upper and lower jaws (film size 3×4 cm^2).
  - Two exposures for maxillary canines (film size 2×3 cm^2); the distal surface of the lateral incisor is also covered.
  - One exposure for maxillary central incisors (film size 2×3 cm^2); mesial surfaces of lateral incisors are also covered.
  - Two exposures for mandibular canines (film size 2×3 cm^2).
  - One exposure for mandibular incisors (film size 2×3 cm^2).
- Procedure:
  - Patient sits upright in a chair.
  - The film is positioned in its holder in the open mouth of the patient under visual control.

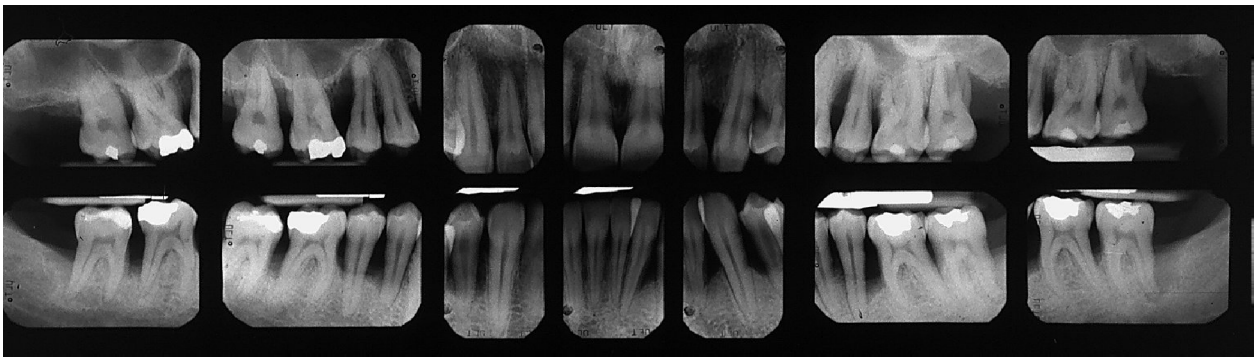


Fig. 7.**13** X-ray survey: 14 periapical radiographs

- A cotton roll is placed on the dental arch of the opposite jaw in order to fix the film holder when the mouth is closed.
- The long cone of the X-ray machine is adjusted, by reference to the adjustment device on the film holder, until it is perpendicular to the film.
- The film is exposed for a time that depends on film type and tooth group.

➤ Alternatively, especially for lateral teeth, *vertical bitewing exposures* may be possible (this requires a special film holder, e.g., VIP-2, Up-Rad Corp., Fla., USA):
- For bimaxillary assessment of bone loss up to about half of the root length.
- Advantages:
  - The film is not bent, or is only minimally bent (related to the hard palate or floor of the mouth).
  - Considerable reduction in the number of exposures.
  - Film holders can be individualized by occlusal impressions made with self-curing resin, which allows standardization of exposures to a large extent; e.g., for follow-up documentation in clinical studies.
- Disadvantage: anterior regions can usually not be assessed because of considerable overlap.

➤ *Bisecting-angle technique* (Fig. 7.**12 b**) is generally not recommended:
- The positioning of the film is uncertain; it is fixed by the patient's index finger.
- Film distortion is uncontrolled.
- Cone adjustment is uncertain.
- *Note:* Nowadays, even for endodontic exposures complicated with rubber dam, rubber dam clamps, and inserted root canal instruments, the paralleling technique is usually possible using special film holders.

➤ The periapical survey may be supplemented by *horizontal bitewings* to assess interproximal carious lesions. *Note:* In children and adolescents, bitewings may be sufficient to diagnose incipient bone loss at the alveolar crest.

➤ Direct *digital radiography* to capture a radiographic image: intraoral detector (luminescence radiography with a scanner) or sensor chip (CCD):
- Advantages:
  - Reduced radiation dose.
  - Digital image manipulation is possible.
  - Possibility of qualitative and quantitative subtraction radiography; computer-assisted digital image analysis (CADIA).

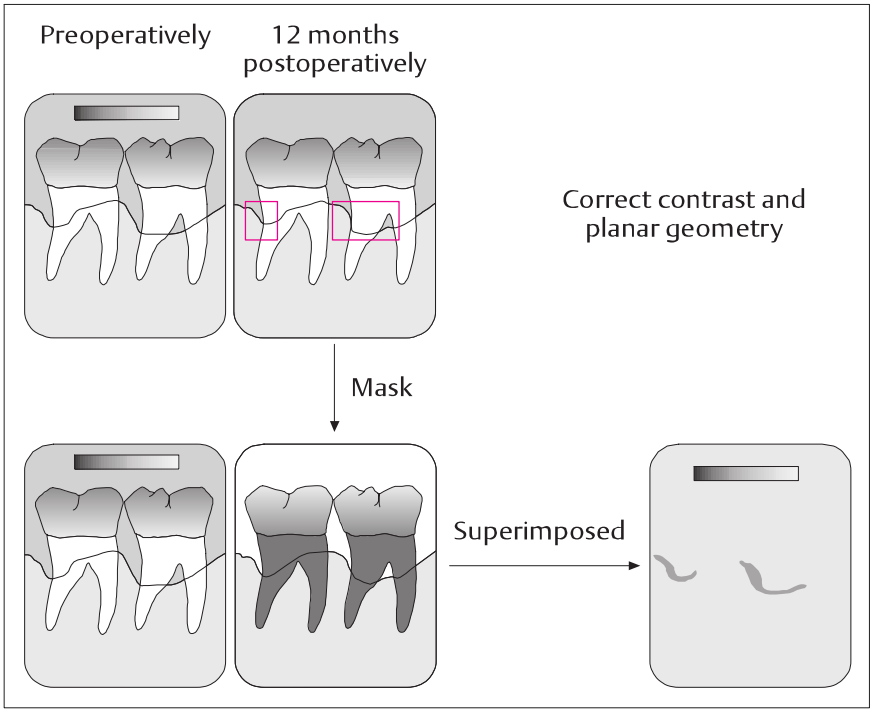


Fig. 7.**14** Principles of subtraction radiography. In this example we see two digital or digitized radiographs with acceptable radiation geometry, showing a particular region of interest and taken 12 months apart. Differences in contrast and brightness can be compensated for by appropriate software, as can small distortions of the planar geometry (some bending of the film). A mask (negative) is made of the postoperative radiograph and the two radiographs are superimposed. Leaving out the background noise, the bony alterations on the distal aspects of both molars and in the furcation area of the first molar now become visible

- Disadvantages:
  - Sensor field is rather small (2×3 cm^2), so more exposures are usually necessary for a whole survey.
  - Fraudulent digital manipulations are possible.

➤ Subtraction radiography (Fig. 7.**14**) presupposes a high level of standardization with regard to radiation geometry, brightness, and contrast. The last two can be adjusted using appropriate software.

Computed Tomography

➤ High-resolution computed tomography:
- Advantages: can show slices of the periodontium in two planes, e.g.:
  - Furcation areas
  - Intrabony pockets
  - Bony dehiscences
- Disadvantages:
  - Very expensive method
  - High radiation dose
  - Artifacts arising from metallic restorations
- Presently mainly employed in preimplantation diagnosis.

Intraoperative Diagnosis of Defect Morphology

General Remarks

➤ When expensive and complicated regenerative measures are contemplated, careful examination of bony lesions together with documentation of the findings is mandatory:
- Both the number of bony walls bordering the periodontal pocket and the morphology of a furcational lesion have prognostic value:
  - The likelihood of bony regeneration increases considerably with the number of bony walls. *Note:* In four-wall defects (i.e., extraction sockets), almost complete bony regeneration can be expected.
  - If conditions suggest there is little hope of success, the treatment plan should be revised. *Note:* The final assessment of defect morphology can only be done intraoperatively.
- The dentist has an obligation to maintain proper documentation both for the patient and for the insurance company or public health service that bears the treatment cost.

Osseous Defects

➤ Infrabony defects (Fig. 7.**15**) and furcation involvements may be preliminarily diagnosed on radiographs (OPT, periapical radiograph). The definitive *classification of bony defects*, however, can only be done during surgery:
- Definition: the bottom of the pocket is apical to the alveolar crest.
  - Three-wall bony defect: interproximal bony pocket where the buccal and lingual bony walls and proximal bone at the neighboring tooth are preserved. A special case of infrabony defect, also termed "intrabony defect."
  - Two-wall bony pocket: interproximal bony pocket with loss of either the buccal or lingual bony wall.

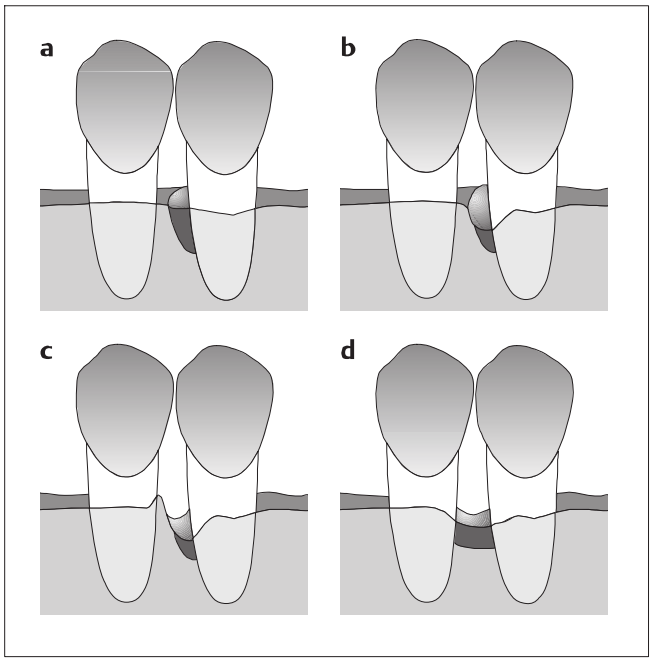


Fig. 7.**15**
a Three-wall bony pocket.
b Two-wall bony pocket. The defect is bordered by either one buccal or lingual wall and one proximal bony wall.
c Mostly one-wall bony pocket, but note that apically there are several walls.
d Interdental crater with bony walls buccally and lingually

- One-wall bony pocket: interproximal bony pocket with loss of both buccal and lingual bony wall.
- Interdental crater: buccal and lingual bony walls are preserved while bony support at the neighboring tooth has been lost.

– Combinations are frequently seen:
- Three-wall bony pocket at the bottom of the lesion
- Two-wall component in the middle of the lesion
- One-wall component more coronal

– The circumference of the bony lesion is estimated, e.g., in steps of 30°.

➤ The number of walls of bony pockets can also be documented in lesions that are not located interproximally.

Furcation Involvement

➤ Intraoperative revision of furcation involvement (Figs. 7.**16**, 7.**17**):
– Furcation depth
– Width of furcation entrance
– Height of furcation entrance, e.g., classification according to Tarnow and Fletcher (1984):
- Class A: <3 mm
- Class B: 4–6 mm
- Class C: >6 mm

– Height of the root trunk
– Height of interdental bone

Fig. 7.**16** Intraoperative diagnosis of bony lesions in the furcation area. Assessment of the width of the furcation entrance at the level of the alveolar crest, as well as height and depth of the furcation, height of the root trunk and distance between cementoenamel junction and bottom of the defect.

– Special circumferential bony pockets with indirect furcation involvement

Advanced Diagnostic Techniques

General Remarks

➤ In some problematic cases *special tests* may be required:
– Aggressive forms of periodontitis
– Refractory cases without acceptable therapeutic response

➤ *Note:* The amount of information gained must be in reasonable proportion to the cost in time and resources.

Diagnostic Test Systems

➤ Beyond the clinical and radiographic findings, diagnostic tests should provide the doctor with further information about, e.g.:
– The nature of the infection
– Prognosis
– Treatment outcome.

➤ Special diagnostic tests might answer the following questions:
– Is destructive periodontal disease likely to develop?
– Is the existing periodontal lesion active, i.e., progressive, or is it stable?
– Is the periodontitis aggressive (rapidly progressive), or is it chronic (slowly progressive)?

➤ Conventional diagnostic measures frequently lead to simultaneous over- and undertreatment. Ideally, diagnostic tests should enable the dentist to give appropriate therapy.

➤ Development of a diagnostic test requires a so-called *gold standard*, which definitively signals the presence or absence of disease:
– The gold standard is usually very invasive
- e.g., histological evidence of attachment loss following extraction of the tooth.

– In clinical practice, the gold standard should be replaced by noninvasive parameters, i.e., validated test parameters. For example, a clinically determined attachment loss or radiographically observed bone loss occurring in a relatively short

Patient: N. L.		*: 05/18/1936			Date: 05/20/2000			Tooth: 26		
Strategic importance:	high									
Sensitivity:	+									
Endodontic situation:	./.									
Mobility:	I									
Restorative situation:	sufficient									
		mb		b		db	dl		l		ml
Clinical findings											
	PPD	5	1	6	2	4	4		2		6
	vCAL	3	3	7	2	2	2		0		4
	hCAL			Ø			2				Ø
	Furcation degree			FIII			FI				FIII
Radiographic findings											
	Bone loss (% root length)	40	0		0	30	15				40
Intraoperative findings											
	Bone height										
	CEJ-BD	3.5	3.5	5.5	2.5	2.5	2		1.5		5
	CEJ-AC	3.5	3.5	7.5	2.5	2.5	2		1.5		5.5
	3-wall comp.			2							0.5
	2-wall comp.			0							
	1-wall comp.			0							
	Furcation										
	Depth			Ø			2.5				Ø
	Degree revised			FIII			FI				FIII
	Width			2			2				2.5
	Height			2			1.5				2.5
	Root trunk			2			2.5				2

Fig. 7.**17** Documentation chart for bony periodontal lesions at a first molar in the maxilla *mb* mesiobuccal; *b* buccal; *db* distobuccal; *dl* distolingual; *l* lingual; *ml* mesiolingual; *PPD* periodontal probing depth; *vCAL* vertical clinical attachment level; *hCAL* horizontal clinical attachment level; *FI–III* furcation degrees I–III; *CEJ–BD* distance from cementoenamel junction to bottom of defect; *CEJ–AC* distance from cementoenamel junction to alveolar crest

time may signal active destruction of the supporting apparatus of the tooth.

- Attention should be paid to the following problems:
  - The real attachment level cannot be determined by clinical probing.
  - The measurement error of periodontal probing is about 0.5–1 mm.
  - Qualitative or quantitative subtraction radiography (to observe bone loss/gain, for example) presupposes a very high level of standardization of exposures.

➤ Using these inferior *surrogates* (as compared to the true, histological gold standard), diagnostic tests have been developed which are based on completely different test systems, which include:

- potential periodontal pathogens;
- local mediators of the inflammatory and immunological host response;
- metabolic products of host and bacteria;
- deficiencies and peculiarities of the immune system;
- genetic polymorphisms.

➤ The test parameters of a diagnostic test can be estimated in a screening experiment (Fig. 7.**18**):

- Sensitivity: the conditional probability that a diseased individual is correctly identified by the test (true-positive result)

	Positive test result	Negative test result
Disease present	a	b
Disease absent	c	d

Sensitivity (rate of true-positive test results) **= a/(a+b)**

Specificity (rate of true negative test results) **= d/(c+d)**

Fig. 7.**18** Diagnostic test parameters are usually derived from a screening experiment. The sensitivity of the test is calculated as the proportion of true-positive results (as compared with the gold standard), while the specificity is the proportion of true-negative results

- Specificity: the conditional probability that an individual not affected by the disease is correctly identified by the test (true-negative result)

➤ The usefulness of a diagnostic test in the clinical setting depends not just on these test parameters, but more essentially on three others:
- The prevalence of the disease in the population
- The importance of the disease for the individual and for the population at large
- The costs of the test

➤ For a life-threatening disease with a high risk of transmission, very high sensitivity (e.g., 99%), and even higher specificity (e.g., 99.9%) is required for a diagnostic test. *Example:*
- The prevalence in a certain population of individuals infected with a certain virus is 0.1%.
- Only 2×10^{-6}% of negatively tested individuals are actually infected by the virus. This *false-negative rate* can be ignored.
- On the other hand: about 50% of individuals who test positive are actually not infected–a very high *false-positive rate*!
- Interpretation: the test is still useful, since virtually all individuals infected with the virus are identified:
  - Preventive and therapeutic measures can be implemented immediately.
  - The population as a whole can be protected.
- Consequences: persons who test positive must be retested by a better, usually more expensive test, to reduce the false-positive rate.

➤ For a non-life-threatening and common disease such as active periodontitis, which can be treated by simple measures such as subgingival scaling, diagnostic test parameters are not so demanding. A moderate sensitivity of about 70% and a relatively high specificity of about 90% may result in a useful test if the costs of the test are not exorbitantly high. *Example:*
- Suppose the prevalence of active periodontitis in a population amounts to 5%.
- 73% of positive test results would relate to periodontally stable individuals/tooth surfaces: this would give a relatively high false-positive rate.
- 1.7% of negative test results would relate to periodontally active patients/tooth surfaces: this is a relatively low false-negative rate.
- Consequences: the test would lead quite frequently to (over-)treatment of conditions that are actually stable. On the other hand, virtually every active lesion would be identified.

Table 7.**2** Evidence for pathogenetic role of potential periodontal pathogens. (Modified from Haffajee and Socransky 1994)

Very strong	Strong	Moderate	Insufficient
Actinobacillus actinomycetemcomitans	*Prevotella intermedia*	*Streptococcus intermedius*	*Selenomonas* spp.
Porphyromonas gingivalis	*Campylobacter rectus*	*Prevotella nigrescens*	Gram-negative enterobacteria
Tannerella forsythia	*Eubacterium nodatum*	*Micromonas micros*	Staphylococci
Invasive spirochetes in necrotizing ulcerative gingivitis/periodontitis	*Treponema denticola*	*Fusobacterium nucleatum*	*Campylobacter gracilis*
		Eubacterium spp.	
		Eikenella corrodens	

Table 7.**3** Comparison of microbiological tools used in periodontal diagnosis

Method	Detection limit	Expenditure	Comments
Culture • Full media • Selective media	 10^4–10^5 10^2–10^3	One week to several weeks	• Overview of the total viable, cultivable flora • Identification of uncommon bacteria • Antibiotic sensitivity testing possible
Immunological tests	10^2–10^4	Chairside up to 1 day	• Usually only limited number of target bacteria • Identification of living and dead bacteria
Enzyme tests (BANA, other oligopeptides)	10^4	Chairside	• Nonspecific for *P. gingivalis*, *T. forsythia*, *T. denticola* • *Capnocytophaga* spp. and certain *Actinomyces* spp. are also BANA-positive
Molecular biological tests • DNA probes • PCR	 10^2 (1–)10	 Days Hours	• Very specific oligonucleotide probes for 16S-rRNA • Commercially distributed tests for a limited number of bacteria • Dead bacteria can be identified

  - The consequences of overtreatment (unnecessary costs) are outweighed by the risks of possible undertreatment (irreversible loss of tooth-supporting structures).

➤ Traditional clinical parameters such as redness of the gingiva, bleeding on probing, purulent exudate, and probing pocket depth, are only moderately specific (≥70 %) and not very sensitive (≥30 %). There is therefore a great desire to develop test systems that have the potential to gain more information.

Microbiological Tests

➤ A microbiological approach to diagnosing periodontitis appears reasonable because of the infectious nature of the disease and because a limited number of potential pathogens are involved (Table 7.**2**).

➤ Table 7.**3** gives an overview of different tests for the microbiological diagnosis of periodontal disease.

➤ In 1683, Anton van Leeuwenhoek described various bacterial morphotypes in dental plaque and their motility for the first time. At the end of the nineteenth century, in Robert Koch's laboratory, W.D. Miller was the first who cultivate dental microorganisms. Over the past 20 years, more sensitive and specific investigation tools have been developed.

➤ *Microscopic techniques* (darkfield and phase contrast microscopy, gram preparation):

  - Gram-negative bacteria increase drastically in numbers and proportions in pathologically deepened pockets.
  - There is also a considerable increase in motile bacteria (motile rods and spirochetes):
    - The ratio of nonmotile to motile organisms in healthy conditions is about 1:40.
    - In periodontitis it rises to 1:3 or up to about 1:1.
  - However, bacteria cannot be differentiated at the species level.
  - Microscopic techniques can be useful in patient motivation, *but not* in bacteriophobic patients.

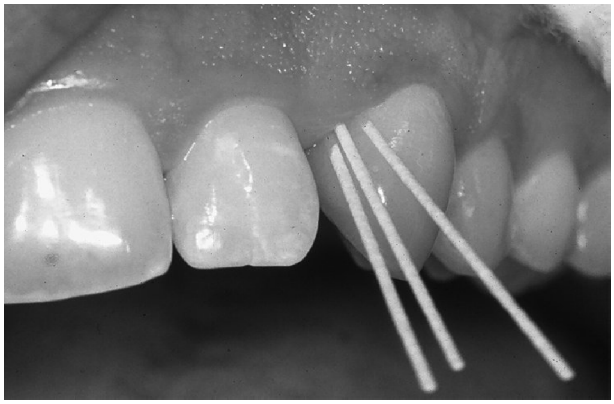


Fig. 7.**19** A subgingival sample is obtained by inserting three endodontic absorbent paper points into the pocket. They are left in place for about 10 seconds and then immediately transferred into a suitable prereduced transport medium

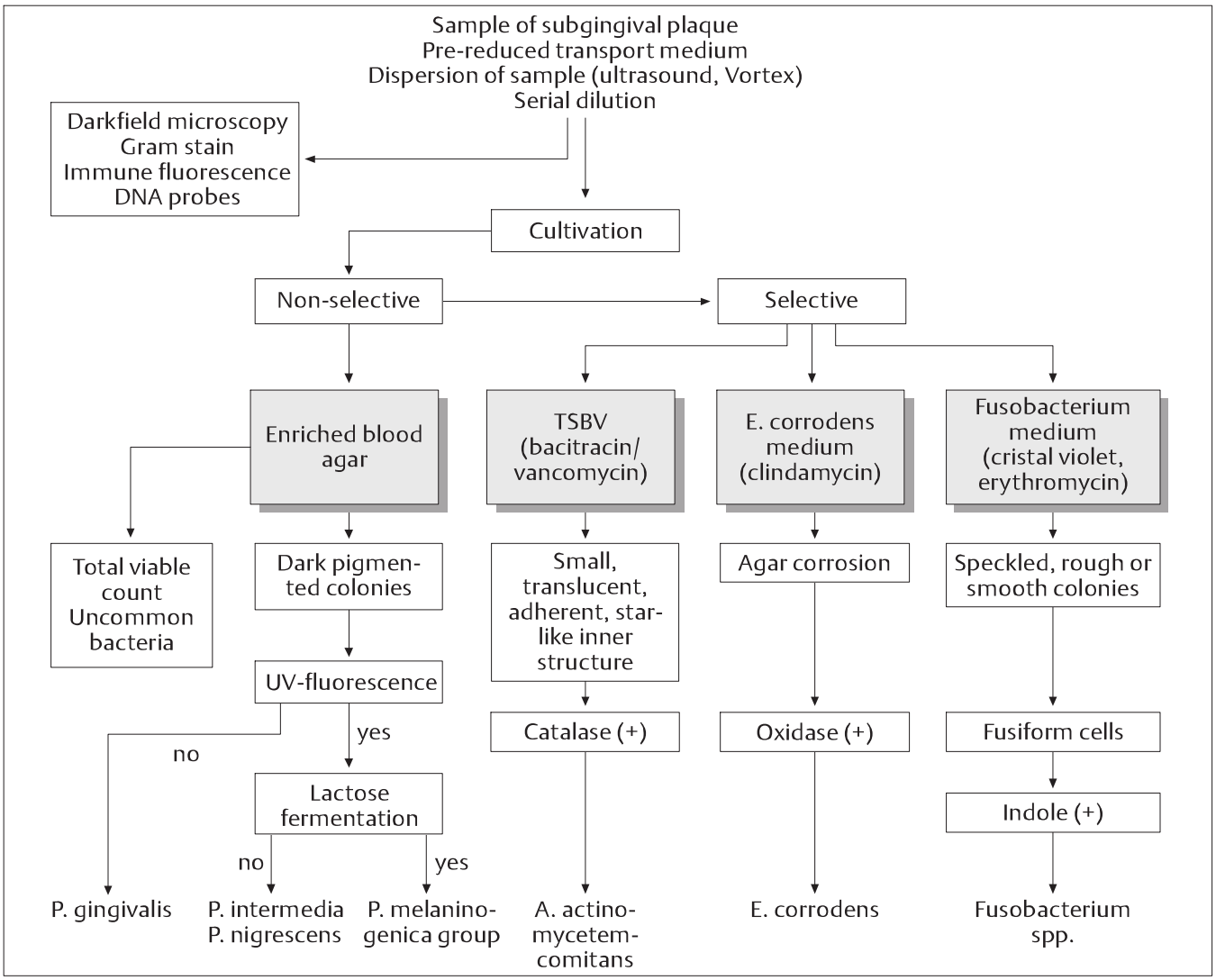


Fig. 7.**20** Cultivation of a sample of subgingival plaque and preliminary identification of potential gram-negative periodontal pathogens. The total count of viable colony-forming units is determined on nonselective enriched blood agar taking account of the dilution factor. Some dark-pigmented colonies can be differentiated by their fluorescence capacity under long-wave UV light (*P. gingivalis* or *P. intermedia*/*P. nigrescens*, *P. melaninogenica* group). Further differentiation is possible by lactose fermentation. Pink or opalescent, round, convex, colonies of fusiform bacteria are probably *T. forsythia* (subcultivation and definitive differentiation on blood agar containing *N*-acetyl-muraminic-acid is necessary), while transparent, flat, spreading colonies may be *Campylobacter* spp. (subcultivation and definitive differentiation on formate-fumarate agar). Selective media for *A. actinomycetemcomitans*, *E. corrodens*, and *F. nucleatum* allow preliminary identification, enumeration, subcultivation, and definitive identification of these microorganisms. (Adapted from Slots 1986)

- *Bacterial culture:*
  - Very time-consuming and expensive.
  - Allows identification of established periodontal pathogens in particular.
  - In addition, unusual bacteria such as pseudomonads, enteric rods, staphylococci, *Candida* spp. etc., may be identified.
  - Antibiotic susceptibility testing can be conducted.

- Procedure:
  - Remove all supragingival deposits.
  - Sample subgingival plaque with endodontic absorbent points (ISO 30 or 35), which are inserted into the pocket for about 10 seconds (Fig. 7.**19**).
  - Transfer the sample into prereduced transport medium (e. g., Port-A-Cul) and send it

to the microbiological laboratory immediately.
- There the sample is treated with ultrasound to disperse it in the transport medium.
- It undergoes serial dilution and aliquots are streaked out onto various agar plates:
  - Nonselectively on enriched blood agar
  - Selectively on media containing antimicrobial dyes and/or antibiotics that support growth of certain species, such as *A. actinomycetemcomitans*, *E. corrodens*, *Capnocytophaga* spp., *Fusobacterium* spp., *Actinomyces* spp., streptococci etc., while suppressing growth of competing microorganisms.
- The plates are incubated under strictly anaerobic and/or aerobic conditions.
- After 7–10 days the plates are inspected. The total number of colonies and the proportions of particular colony-forming units (CFUs) are determined. After allowing for the dilution factor, the number of CFUs per sample can be calculated (Fig. 7.**20**).
- Individual colonies are picked, and subcultivated for further seven days.
- The pure cultures are identified by means of numerous tests:
  - Colony morphology
  - Gram preparation
  - Biochemical tests
  - Gas chromatography of metabolic end products, etc.
- Testing of the sensitivity of the cultures to various antibiotics:
  - Agar diffusion test, E-test
  - Agar dilution test
  - *Note:* Minimal inhibitory concentrations may be 100 to 1000 times higher for bacteria organized in a biofilm than for the same bacteria living in planktonic cultures.

➤ *Immunological methods* are based on monoclonal antibodies or species-specific polyclonal antisera:
- *Indirect immune fluorescence:*
  - The sample of subgingival plaque is diluted and heat-fixed on a slide.
  - Monoclonal mouse antibodies or species-specific polyclonal rabbit antiserum is added, which bind at the respective surface antigens of the bacteria.
  - Anti-mouse or anti-rabbit IgG antibodies are added which are conjugated with a special fluorescence dye (fluorescein isothiocyanate).
  - Fluorescing cells are identified and counted in a darkfield microscope.
- *Latex agglutination:*
  - Species-specific antibodies are bound to latex particles.
  - When the plaque sample is added, the specific antigens bind to the latex particles.
  - Subsequent agglutination of the latex particles allows a visual assessment.
- *Enzyme immunoassay* (EIA, Fig. 7.**21**):
  - Species-specific antibodies are bound to a membrane in a reaction well.
  - Specific antigens in the plaque sample bind to these antibodies.
  - After the addition of another antibody, which is conjugated to an enzyme, the

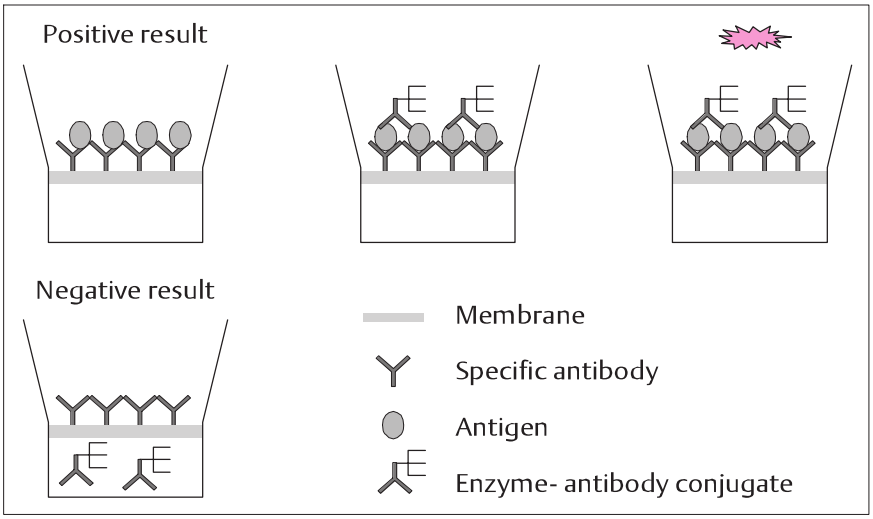


Fig. 7.**21** Miniaturized immune assay for chairside identification of *A. actinomycetemcomitans*, *P. gingivalis*, and *P. intermedia* in plaque samples. Specific antibodies are bound to a membrane. After adding the sample, specific bacteria are bound to antibodies. A further specific antibody which is conjugated to an enzyme is added. The test system can be developed with a special substrate which yields a characteristic dye after enzymatic cleavage

system is "developed" using a suitable substrate.
- The emerging characteristic dye may be quantified colorimetrically.
- *A. actinomycetemcomitans*, *P. gingivalis*, and *P. intermedia* can be identified with this chairside system.

➤ *Enzyme test:*
- *P. gingivalis*, *T. forsythia*, and *T. denticola* possess a trypsin-like peptidase.
- This peptidase hydrolyzes a synthetic peptide, *N*-α-benzoyl-dl-arginine-2-naphthylamide (BANA).
- The cleavage product, β-naphthylamide, is a chromophore and can be visualized by a color reaction.
- However, beneficial bacteria as *Capnocytophaga* spp. and certain *Actinomyces* spp. are also BANA-positive.

➤ *Molecular biological methods:*
- Species-specific enzyme- or radiolabeled oligonucleotide sequences (DNA probes) bind to complementary sequences of bacterial DNA:
  - Hypervariable sequences of 16S rRNA, which are known for a large number of species, are very suitable for constructing species-specific DNA probes.
  - Oligonucleotides consisting of 24–30 base pairs are either radioactively or enzymatically conjugated, giving fluorescence or chemiluminescence (digoxigenin-labeled).
  - In contrast to these oligonucleotide probes, whole genomic or cloned DNA probes more frequently show cross-reactions.
- Procedure:
  - The plaque sample is dispersed in transport medium.
  - Enzymes and detergents are added to lyse cells.
  - NaOH is added to denature the bacterial DNA.

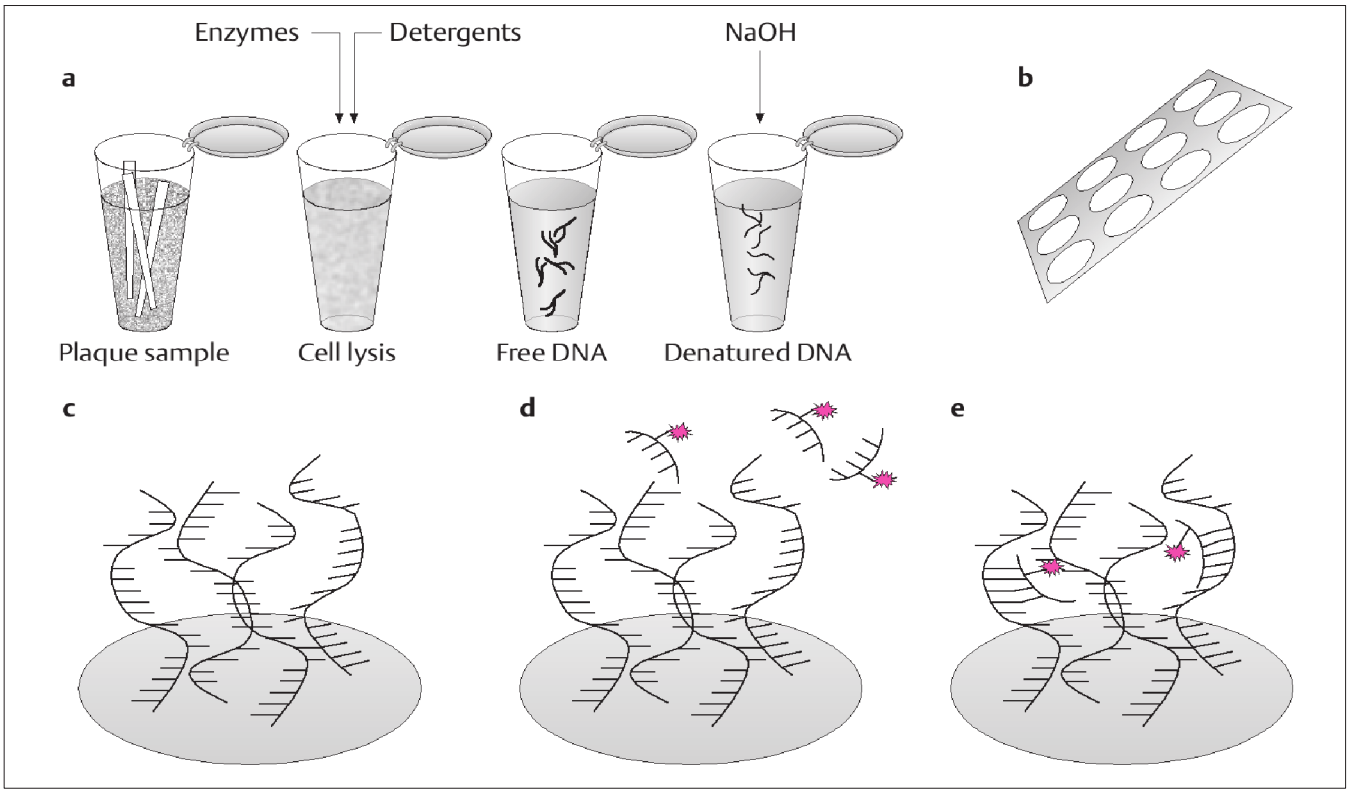


Fig. 7.**22** Principle of DNA probes.
a Sampling and denaturation of bacterial DNA, which is bound to special filter material (**b**).
c High magnification of one spot showing floating, single-stranded DNA.
d Species-specific, labeled DNA probes are added.
e Probes bind only to target zones of homologous bacterial species. Labeling allows semiquantitative assessment. (Adapted from Murray and French 1989)

- DNA probes bind only to complementary fragments (Fig. 7.**22**).

– DNA probes for *P. gingivalis*, *P. intermedia*, *A. actinomycetemcomitans*, *E. corrodens*, *F. nucleatum*, *C. rectus*, *T. forsythia*, and *T. denticola* are commercially available.

– *Polymerase chain reaction* (PCR): a pair of oligonucleotide primers is used to amplify a small sequence of genomic DNA several million times, thus making it detectable (Fig. 7.**23**). Primers usually consist of between 18 and 28 bases.
  - Various sequences of genomic DNA can be amplified, e.g., species-specific 16S rRNA, sequences in the region of the leukotoxin gene of *A. actinomycetemcomitans* (Fig. 7.**24**) etc.
  - The sensitivity of PCR is very high.

Markers of Specific and Nonspecific Host Response

➤ Results of serological examinations may be difficult to interpret:
– In cases of aggressive periodontitis, high antibody titers against periodontal pathogens such as *A. actinomycetemcomitans* always indicate the presence of the microorganism.
– Insufficient production of antibodies may lead to a more generalized form of the disease.

➤ Various biochemical markers of the host response may be identified in inflammatory gingival exudate (Fig. 7.**25**), which can have prognostic significance:
– Markers of the inflammatory reaction
– Host enzymes
– Tissue metabolites

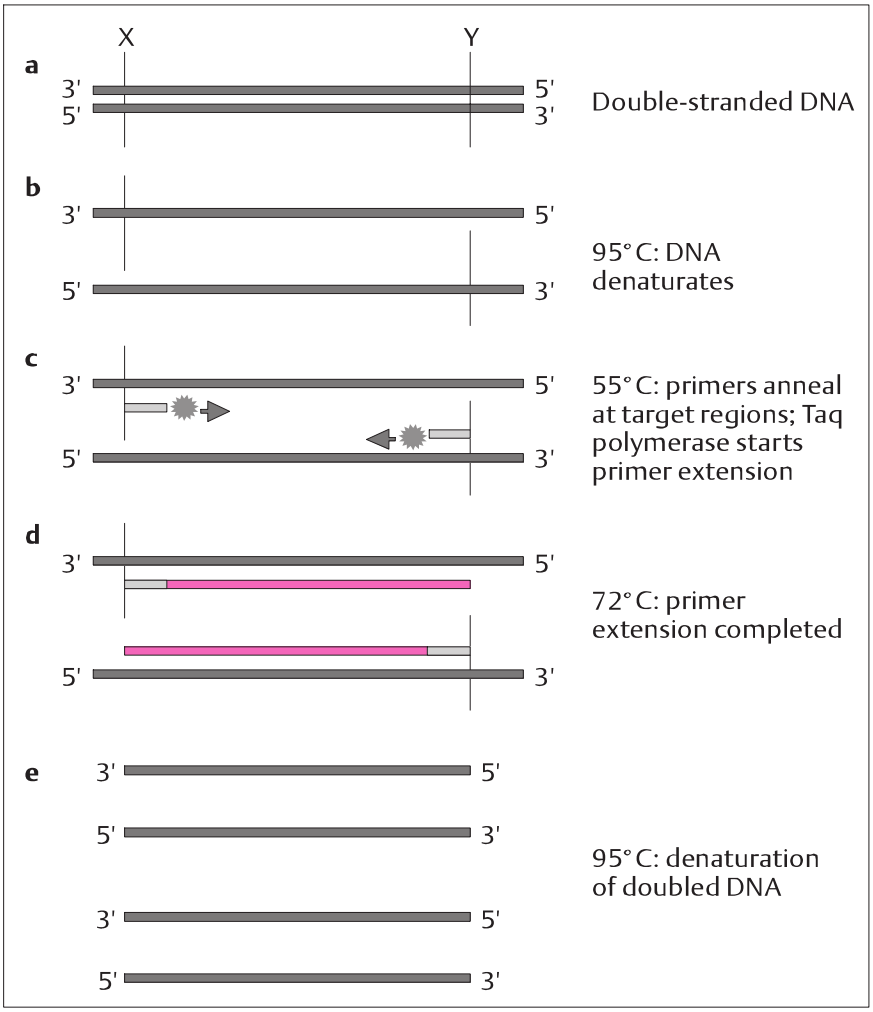


Fig. 7.**23** Principle of polymerase chain reaction (PCR).
a The genomic sequence between X and Y is to be identified by amplification.
b At 95 °C double-stranded DNA denatures, i.e., there are now two single strands of DNA as template.
c After cooling to 55 °C, a pair of specific oligonucleotide primers can anneal (hybridize) to the respective target regions. Heat-resistant Taq polymerase starts primer extension.
d At 72 °C, primer extension leads to double-stranded DNA between the two primers.
e Two double-stranded pieces of DNA can now be further multiplied by repeating the cycle.

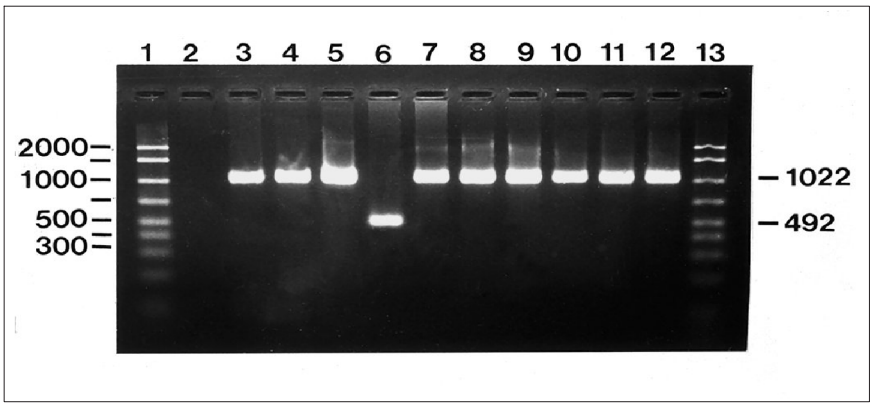


Fig. 7.**24** Gel electrophoresis of amplified sequences after PCR. A sequence within the promoter region of the leukotoxin gene of *A. actinomycetemcomitans* (cf. Fig. 2.**6**) has been amplified and visualized by ethidium bromide. Lanes 1 and 13 represent DNA size markers, lane 2 is a negative control (water). The analysis revealed two sequences with different sizes. In most species, 1022 base pairs were amplified (lanes 3–5, 7–12). One isolate (lane 6) had a deletion of 530 base pairs in the promoter region of the leukotoxin gene

- Markers of the inflammatory reaction:
  - Monocytes of individuals susceptible to periodontitis respond to LPS of gram-negative bacteria with two- to three-fold increased secretion of PGE_2:
    - PGE_2 may be found in gingival exudate and connective tissue.
    - It is strongly associated with local progression of periodontitis.
    - An EIA for PGE_2 in gingival exudate is available.
    - Similar observations have been made in relation to the proinflammatory cytokines IL-1α and IL-1β, IL-6, and TNF-α, which can be detected by EIA in increased concentrations in gingival exudate, especially in refractory cases.
    - *Note:* A postulated *hyperreactive phenotype* of macrophages may be essentially controlled by genetic factors, but may be modulated by acquired and behavioral factors.
  - The *acute-phase reactants* C reactive protein (CRP), α_1-macroglobulin, α_2-proteinase inhibitor, and transferrin and lactoferrin do not appear to be suitable for the identification of active periodontal lesions.
- Host enzymes:
  - *Matrix metalloproteinases*, such as collagenase and gelatinase, play an important role in periodontal tissue remodeling and turnover:
    - Neutral proteases may be nonspecifically detected in gingival exudate by a colorimetric assay.
    - A soluble, colored collagen fragment is cleaved enzymatically from a nonsoluble dye-and-collagen conjugate.
  - Similar methods have been described for the detection in gingival exudate of *lysosomal enzymes* derived from polymorphonuclear granulocytes:
    - Elastase
    - Cathepsins
    - β-Glucuronidase
  - *Alkaline phosphatase* is released by osteoblasts, fibroblasts, and neutrophilic granu-

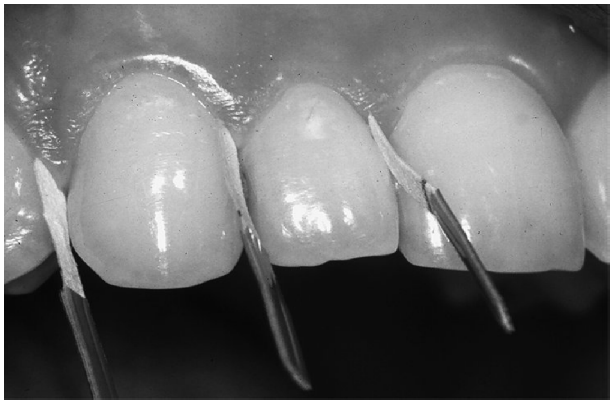


Fig. 7.**25** Sampling of inflammatory gingival exudate at the mesial aspects of teeth using filter paper strips placed at the orifice of the sulcus/pocket

locytes. This enzyme can be detected in gingival exudate by chemiluminescence
  - *Aspartat aminotransferase* (AST) is released only after cell death. Increased concentrations of AST in gingival exudate (>800 mIU) have been associated with active periodontal destruction. A test kit has been developed.
- Products of tissue metabolism:
  - Glycosaminoglycans are polysaccharide compounds which are bound to tissue proteoglycans. The occurrence especially of *chondroitin-4-sulfate* and *chondroitin-6-sulfate* in gingival exudate has been correlated with gingival inflammation.
  - The amino acid *hydroxyproline* is a major component of collagen. Since it is detectable in gingival exudate even in gingivitis, it appears not to be suitable as marker of periodontal destruction.
  - The same holds true for *fibronectins*, a heterogeneous group of glycoproteins in blood and connective tissue which play an important role during inflammation and wound healing.
  - Further connective tissue proteins as osteonectin, osteocalcin, or type-I collagen peptides, carboxyterminal type-I propeptide, and aminoterminal type-III propeptide, and others can be detected in gingival exudate. Associations with periodontal destruction are currently under investigation.
- Critical remarks:
  - Some biochemical markers in gingival exudate appear to be associated with periodontal activity.
  - Others are highly correlated with clinical inflammation.
  - *Note:* At present, biochemical markers are not suitable for periodontal diagnosis:
    - Possible tests yield little or no additional information.
    - High costs are incurred if multiple sites are tested
    - Thus, most of the test systems developed to date have not been adopted in daily practice.

Human Genetic Tests

- Genetic markers:
  - Aggressive forms of periodontitis that in themselves are rare are more frequent in some families:
    - There may be a *single genetic locus* with an autosomal dominant inheritance that is responsible for increased susceptibility to early-onset (aggressive) periodontitis.
    - Alternatively, several *modifying genes* may control an individual's host response (e.g., IgG2 production on infection with gram-negative bacteria of dental plaque).
    - There may be a combination of both.
- In many cases of aggressive periodontitis, functions of the polymorphonuclear granulocytes are impaired, while concomitantly the monocytic response to LPS is enhanced:
  - The relevant gene loci (Table 7.**4**) may be suitable for the development of human genetic tests for the individual's determination of periodontitis susceptibility.
- *Polymorphisms of the FcγRIIa receptor* (chromosome 1q21-q23):
  - FcγRIIa on monocytes/macrophages and neutrophilic granulocytes is the only receptor of the FcγR family that binds effectively to IgG2.
  - Variants of the receptor differ at position 131: histidine (H) or arginine (R):
    - H131 homozygotes are not at increased risk of aggressive periodontitis.

Table 7.**4** Genetic variants, which have been associated with an increased susceptibility for periodontitis. (After Page et al. 1997)

Genetic variant	Gene locus, chromosome
Functional anomalies of phagocytes	
Variable functions of monocytes/ macrophages	
Reduced production of IgG2	6
FcγRIIa receptor	1q21-q23
TNF-α polymorphism	6
IL-1 gene cluster	2q13-q21
Cyclooxygenase-1	9q32,33
Cathepsin C	11q4

- R131 homozygotes may have increased susceptibility to infection with *Neisseria meningitidis* and *Haemophilus influenzae* because of the weak binding of the receptor to IgG2. It is conceivable that the risk of aggressive periodontitis is also increased.

➤ *Polymorphisms of the interleukin-1 gene complex* (chromosome 2q13-q21) have been associated with chronic periodontitis in nonsmokers:
- Allele 2 of the IL-1A polymorphism (position −889 or +4845) plus allele 2 of the IL-1B polymorphism (+3954) may result in considerably increased monocytic release of IL-1 after contact with LPS of gram-negative plaque bacteria.
- A commercially available human genetic test is based on an observed association between this haplotype and chronic periodontitis:
  - IL-1-genotype-positive nonsmokers had more severe periodontitis (Fig. 7.**26**).
  - No association between haplotype and periodontitis was found in smokers.
  - Despite intensive supportive periodontal care, IL-1-genotype-positive periodontitis patients may lose more teeth than IL-1-negative patients.
- *Note:* Human genetic tests should never be done for screening but should only be performed in diseased patients.

Bad Breath

➤ The main reason why many patients consult a dentist is for control of actual or imagined bad breath (halitosis).

➤ In most cases, bad breath is due to oral bacteria, which release volatile sulfur compounds, e.g., hydrogen sulfide, dimethyl sulfide, and/or methylmercaptan.

➤ The following ecological habitats should be carefully investigated:
- Periodontal pockets
- Open carious lesions, imperfect margins of restoration
- Dorsum of the tongue
- Tonsils

➤ An olfactory assessment of the expired air may be psychologically problematic and is certainly not very objective.

➤ A gas chromatographic examination of the expired air is reproducible and sensitive (e.g., Halimeter). This measures volatile sulfur compounds nonspecifically.

Diagnosis

General Diagnosis

➤ General diagnosis relates to:
- The *form* of periodontitis (aggressive, chronic)

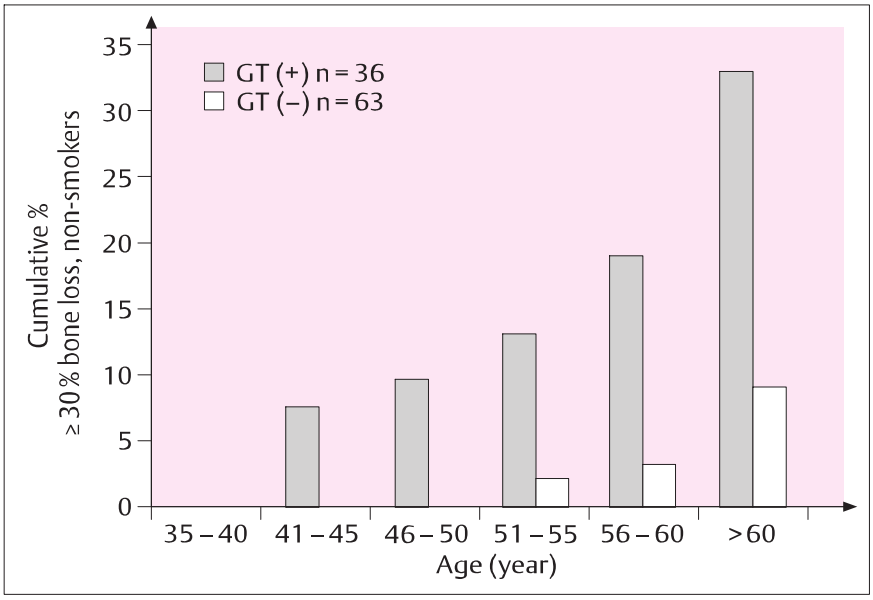


Fig. 7.**26** Cumulative frequencies of nonsmokers with 30 % loss of alveolar bone or more in different age groups. Thirty-six genotype-positive and 63 genotype-negative individuals were examined. (Adapted from Kornman et al. 1997)

– The *extent* of disease (localized, generalized)
– Any modifying *systemic diseases* (e.g., generalized chronic periodontitis modified by type II diabetes mellitus, nonsmoker)
– Any mucocutaneous diseases.

Tooth-Related Diagnosis

➤ In addition, a diagnosis is made for *each tooth*, e.g.:
➤ *Gingivitis* (chronic or acute):
– Periodontal probing depths of 3 mm or less, attachment loss may be present.
– Bleeding upon probing
– Bone loss may be present
➤ Severity of periodontitis may be classified by attachment loss: slight (1–2 mm), moderate (3–4 mm), and severe (≥5 mm). Traditionally, the following classification is used (cf. classification of NHANES III, Chapter 4):
– *Moderate periodontitis:*
• Periodontal probing depth of more than 3 mm and attachment loss
• Bleeding upon probing
• Bone loss up to one-third of root length.
– *Advanced periodontitis:*
• Periodontal probing depth of more than 3 mm and attachment loss
• Bleeding upon probing
• Bone loss of more than one-third of root length, and/or
• Advanced furcation involvement (degree II or III)
➤ *Gingival recession* is present if the attachment loss is greater than the periodontal probing depth
➤ Periodontal abscess

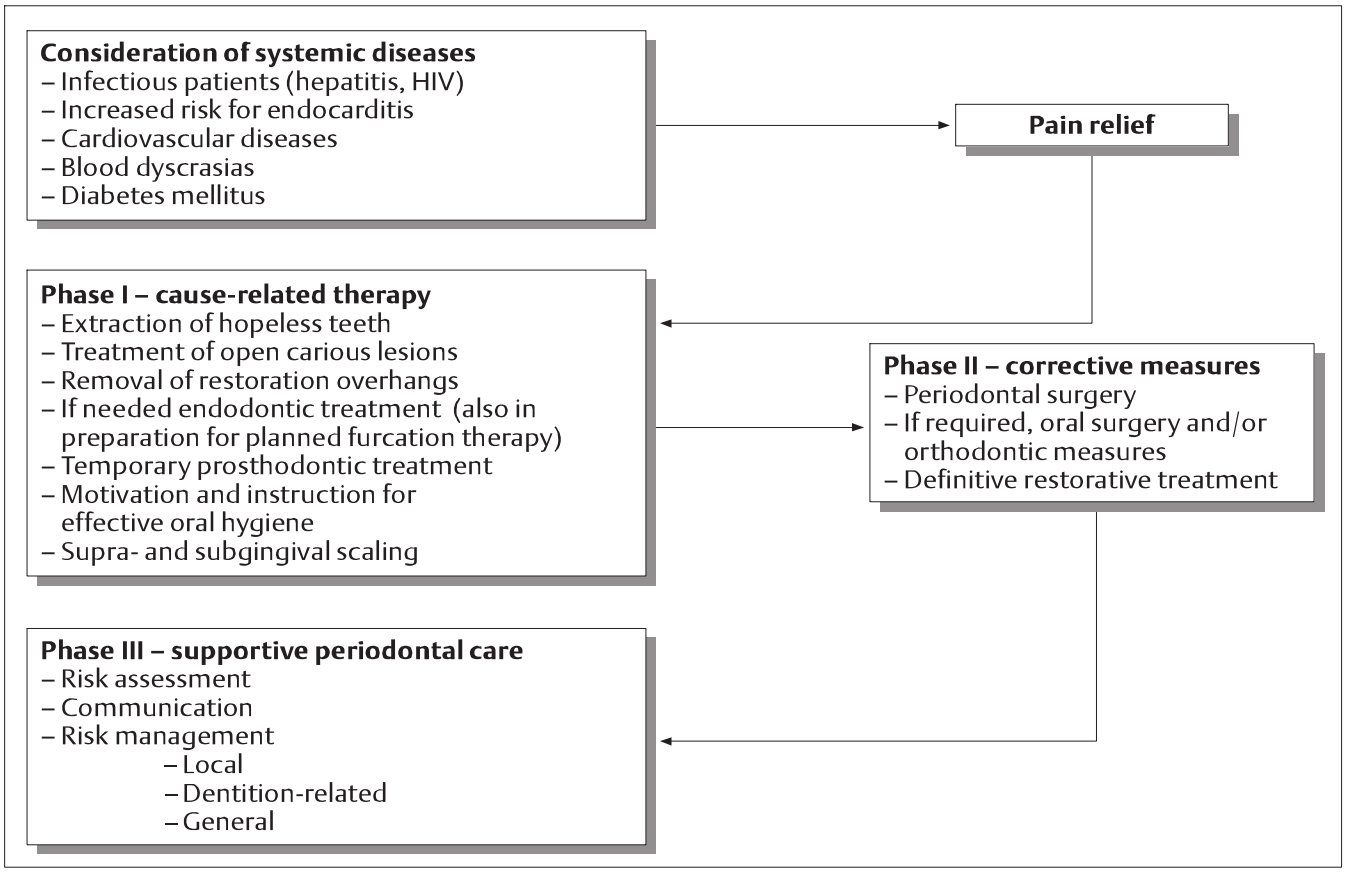


Fig. 7.**27** Phases of comprehensive therapy of a patient with complex periodontal disease

Prognosis

- Combined periodontal–endodontic lesion, etc.
- The individual diagnosis for each tooth is recorded together with its prognosis in the periodontal chart (Fig. 7.**6**):
  - Tooth can definitely be kept.
  - Tooth has questionable prognosis but high strategic importance.
  - Tooth is hopelessly diseased and should be extracted.

Treatment Planning

General Remarks

- Comprehensive treatment of periodontally diseased patients is conducted in well-defined phases (Fig. 7.**27**):
  - Systemic diseases are recorded and carefully considered.
  - Emergency treatment, pain relief.
  - Phase I: cause-related therapy, anti-infectious measures.
  - Phase II: corrective measures to regenerate lost tissue, restorative measures, etc.
  - Phase III: supportive care; risk assessment and risk management.

Case Presentation

- After the medical and dental history have been obtained and a detailed examination and diagnosis carried out, the treatment plan is developed with the patient:
  - The patient is informed about the findings recorded.
  - The dentist asks about the patient's main reason(s) for consultation.
  - The patient's treatment needs in regard to esthetics and chewing comfort are assessed.
  - An optimum treatment proposal is developed.
  - Major aim: to achieve a stable treatment result.
- In addition, at least one medically acceptable minimum solution should be discussed:
  - After briefing about the pros and cons in relation to the time and resources at the patient's disposal, it is the patient who decides which treatment is to be embarked upon.
  - *Note:* Continuous monitoring of advanced periodontal or carious lesions should be avoided during comprehensive therapy.
- A definitive schedule of treatment phases and the emerging costs must be produced.

8 General Medical Considerations

Systemic Phase

General Remarks

➤ Before comprehensive therapy commences, all medical and dental history information is checked for clinical relevance. Any *systemic diseases* must be taken into account during treatment planning:
- To protect the dentist and his/her team from infection (infectious hepatitis due to HBV, HCV; HIV infection; etc.).
- To protect patients at increased risk:
  - Treatment restriction if necessary
  - Drug interactions
  - Contraindications, e.g., allergies
- To achieve an optimum therapeutic result.

Infectious Patients

➤ Relevant pointers in the medical history must be followed up:
- Specific antibody titers
- Membership of a high-risk group

➤ HIV-seropositive patients often suffer from opportunistic infections of the oral cavity, e.g.:
- Necrotizing ulcerative periodontal diseases
- Virus infections: acute herpesvirus infection, hairy leukoplakia (Epstein–Barr virus infection), papilloma virus infection
- Candidiasis; treatment-resistant linear erythema of the gingiva
- Neoplasms, e.g., Kaposi sarcoma

➤ In any of these cases, an HIV test should be arranged.

➤ *Note:* Because even careful history-taking does not always reveal a patient's infectious status, standard precautions for avoiding cross-infection must be adopted with all patients.

Increased Risk of Infective Endocarditis

➤ Infective endocarditis is a life-threatening disease. It is an infection of hemodynamically exposed endocardial structures, especially native or prosthetic cardiac valves. It may occur after transient bacteremia.

➤ In a patient at increased risk of endocarditis (Table 8.**1**), appropriate antibiotic prophylaxis is mandatory even before the periodontal examination. *Note:* Even periodontal probing leads to bacteremia.

➤ Depending on bacterial virulence and the patient's resistance, various forms of endocarditis may be differentiated according to course:

Table 8.**1** Heart anomalies and postoperative conditions which require antibiotic endocarditis prophylaxis, and heart anomalies and postoperative conditions which do not require such prophylaxis

High risk of endocarditis
• Prosthetic cardiac valves • Previous history of infective endocarditis
Moderately increased risk of endocarditis
• Congenital or acquired heart valve dysfunction • Congenital heart disease – Aortic isthmus stenosis – Patent ductus arteriosus – Ventricular septal defects (primum type) – Aortic stenosis – Cyanotic heart disease • Surgically corrected intracardiac lesions with residual hemodynamic abnormalities • Hypertrophic cardiomyopathy • Mitral valve prolapse with regurgitation
No increased risk of endocarditis
• Isolated secundum atrial septum defect • Surgical correction of secundum atrial septum defect, ventricular septum defect, or patent ductus arteriosus after six months and without residua • Previous coronary artery bypass graft • Mitral valve prolapse without regurgitation • Physiological, functional, or innocent heart murmurs • Previous rheumatic fever, Kawasaki disease, or connective tissue disorder without valvular dysfunction • Cardiac pacemakers and implanted defibrillators

- *Acute infective* endocarditis:
  - Septic disease, high fever.
  - Rapid destruction of the endocardium, fatal within less than six weeks.
  - Causative bacteria are *Staphylococcus aureus*, *Streptococcus pneumoniae*, *S. pyogenes*.
- Intermediate *acute-subacute* forms are more frequently caused by enterococci.
- *Subacute* forms:
  - Slight fever, night sweating, weight loss.
  - If left untreated, fatal within six weeks to three months.
- *Chronic infective* endocarditis:
  - Symptoms similar to the subacute form.
  - If left untreated, fatal after more than three months.
- *Note:* Subacute and chronic endocarditis are usually caused by viridans streptococci.

➤ In persons with *congenital* and *acquired heart anomalies* and *heart failure* the risk of infective endocarditis is considerably increased. It is highest in patients with *prosthetic heart valves* and those who have a *previous history of endocarditis*.
- Predisposing conditions:
  - Abnormal blood flow, eddy formation.
  - Congenital or rheumatic valve disease.
- Blood starts to clot on surface alterations of the valves and forms fibrous and thrombotic vegetation (nonbacterial thrombotic endocarditis).
- These appositional thrombi are subsequently colonized during transient bacteremia.

➤ Lesions in areas of high turbulence are particularly prone to bacterial colonization:
- Atrial surface of the mitral valve
- Ventricular surface of the aortic valve

➤ Bacteria have different affinities to the thrombus, which is primarily bacterium-free:
- Viridans streptococci, enterococci, *S. aureus*, *S. epidermidis*, and *Pseudomonas aeruginosa* adhere better than *Escherichia coli*, *Klebsiella pneumoniae*, and *A. actinomycetemcomitans*.
- Adhesion of the oral streptococci *S. mutans*, *S. bovis*, *S. mitis*, and *S. sanguinis* depends on production of the extracellular polysaccharide dextran.

➤ In addition to *A. actinomycetemcomitans*, other gram-negative bacteria of the oral cavity and upper respiratory tract may cause infective endocarditis: *Haemophilus* spp., *Cardiobacterium* spp., *E. corrodens*, *Kingella* spp., *Capnocytophaga* spp., and *Neisseria* spp.

Table 8.**2** Prescription schedule for endocarditis prophylaxis for interventions in the oropharynx (American Heart Association, Dajani et al. 1997)

No penicillin allergy	Penicillin allergy
• Oral administration of 2 g amoxicillin, one hour before dental procedure	• Oral administration of 600 mg (or 20 mg/kg body weight) clindamycin, one hour before dental procedure or • 2 g (or 50 mg/kg) cefalexin or cefadroxil or • 500 mg (or 15 mg/kg) clarithromycin

➤ *Note:* Bacteremia must be expected during all dental measures which result in bleeding, e. g.:
- placing implants;
- tooth extraction, surgical tooth removal;
- apicectomy;
- suture removal;
- calculus removal;
- local anesthesia;
- periodontal probing.

➤ A patient at increased risk of infective endocarditis requires antibiotic prophylaxis (Table 8.**2**):
- Simplified prescription schedule: the previously recommended additional half-dose after six hours has been dropped.
- *Note:* Appointments should be carefully planned so that as few antibiotic prescriptions as possible are necessary.
- A period of 10–14 days should be kept between appointments to allow recovery of the resident flora.

➤ "Spontaneous" bacteremia in patients with severe gingival inflammation and poor oral hygiene is an additional risk in patients at increased risk of infective endocarditis:
- Healthy periodontal conditions are the best form of endocarditis prophylaxis.
- Preoperative mouth rinsing with chlorhexidine digluconate (0.2%) or application of chlorhexidine onto the dried mucosa reduces bacterial load.

– During periodontal therapy the patient should use a 1 % chlorhexidine gel instead of toothpaste at home in order to shorten the treatment phase.

Further Indications for Antibiotic Prophylaxis

➤ *General rule:* Antibiotic prophylaxis is indicated for:
– any patient with impaired host defenses;
– any surgery with a high rate of associated infectious complications;
– any surgery for which the associated infectious complications are rare (low rate) but serious.

➤ Patients with *organ transplants:*
– Immunesuppressive medication with, e.g., cyclosporine and/or glucocorticosteroids for suppression of T cell functions:
• Generally, consult with the patient's physician, who should make suggestions for antibiotic prophylaxis.
• E.g., for all procedures in which bacteremia is expected: long-term prophylaxis with 1500 mg/day amoxicillin for three days (one day before and two days after the procedure).
• Patients with penicillin allergy: 200 mg doxycycline the day before the intervention, thereafter 100 mg/day; alternatively, erythromycin 1000 mg/day.
– *Note:* During cyclosporine therapy, 25–30 % of patients develop gingival enlargement, which in some cases has to be treated surgically.

➤ *Radiation of the head and neck:*
– Vascular damage with hypovascularity, degeneration of bone marrow, and more or less complete destruction of osteoblasts and osteoclasts lead to a risk of osteoradionecrosis:
• Primary wound closure following, e.g., tooth extraction is mandatory. Antibiotic prophylaxis: amoxicillin 1500 mg/day or clindamycin 600 mg/day on the day of the procedure and up to two days thereafter.
• Larger operations must be carried out in a hospital.
– So far as possible, periodontal treatments should be carried out before radiation. *Warning:* Osteoradionecrosis has been observed after spontaneous exacerbation of chronic periodontitis.

➤ *Antibiotic prophylaxis according to physician's instructions* in:
– Dialysis patients.
– Those with blood dyscrasias.
– Neutropenia, impaired function of polymorphonuclear granulocytes.
– Patients with rheumatic disorders usually have to take corticosteroids: the patient's physician should be consulted about antibiotic prophylaxis.
– Opinions differ regarding antibiotic prophylaxis in patients with joint prostheses. E.g., prescription of 2 g cephalexin one hour before the procedure, and 1 g after four hours (erythromycin or clindamycin in patients who are allergic to β-lactam antibiotics).

Bleeding Disorders, Anticoagulant Therapy

➤ Periodontal treatment of patients with vascular and platelet disorders, *congenital hemophilia*, or, more frequently, those on *anticoagulant medication* (e.g., coumarin derivatives or heparin after myocardial infarction or stroke, or for thrombosis prophylaxis) can only be carried out after consultation with the patient's physician.
– Life-threatening bleeding can occur after any oral surgical procedure:
• Tooth extraction
• Flap surgery
• Gingivectomy
• Calculus removal
– In a patient with an International Normalized Ratio (INR) (based on the patient's prothrombin time) of 3.0–3.4, usually only minor oral surgical procedures can be carried out without any problems.
– If the INR is equal or greater than 3.5, only emergency minor surgical procedures are recommended.
– Comprehensive treatment should ideally be carried out in a few appointments scheduled close together.

➤ *Pharmacological interactions with anticoagulant drugs:*
– Anticoagulant effect is enhanced by, e.g., salicylates and nonsteroidal antiinflammatory drugs, corticosteroids, or certain antibiotics.

- Anticoagulant effect is reduced by barbiturates, resulting in increased risk of thrombosis.

➤ *Note:* Hemophilic patients have a considerably increased risk of hepatitis and HIV infection.

Atherosclerosis, Cardiovascular Diseases

➤ Oral surgical measures represent an increased risk of adverse effects in patients with, e.g., *myocardial infarction*, *angina pectoris*, *heart insufficiency*, *stroke*, or *peripheral vascular disease*.
- The patient's physician should be consulted and all prescribed medications carefully checked.
- No dental treatment for six months after myocardial infarction.
- Diazepam premedication if required; nitroglyceride should be to hand.
- There is no general contraindication regarding local anesthetics with an adrenaline content of up to 1:100 000.
- Only short sessions should be planned.
- In patients with a pacemaker, use of ultrasound, electrosurgical devices, and electric pulp vitality testing are contraindicated.
- *Note:* Patients who have had a myocardial infarction or stroke are frequently on anticoagulant medication.
- Antihypertensive medication should be maintained during dental therapy.

➤ *Calcium antagonists* (nifedipine, diltiazem, verapamil, etc.), frequently prescribed in coronary artery disease, lead in about 20% of cases to gingival enlargement. In some cases surgical correction is required.

Associations Between Periodontitis and Cardiovascular Diseases

➤ In recent years, associations between cardiovascular diseases, especially coronary heart disease and stroke, and certain chronic infections have been reported:
- *Chlamydia pneumoniae:* respiratory infections
- *Helicobacter pylori:* peptic ulcer, gastritis
- Cytomegalovirus
- Dental infections, especially chronic periodontitis:
  - In cross-sectional and longitudinal studies adjusted for established risk factors, the relative risk of coronary heart disease was increased by a factor of 1.2–2 in the presence of periodontitis.
  - DNA of periodontal pathogens *A. actinomycetemcomitans*, *P. gingivalis*, *P. intermedia*, and *T. forsythia* has been detected in atheromatous plaques.

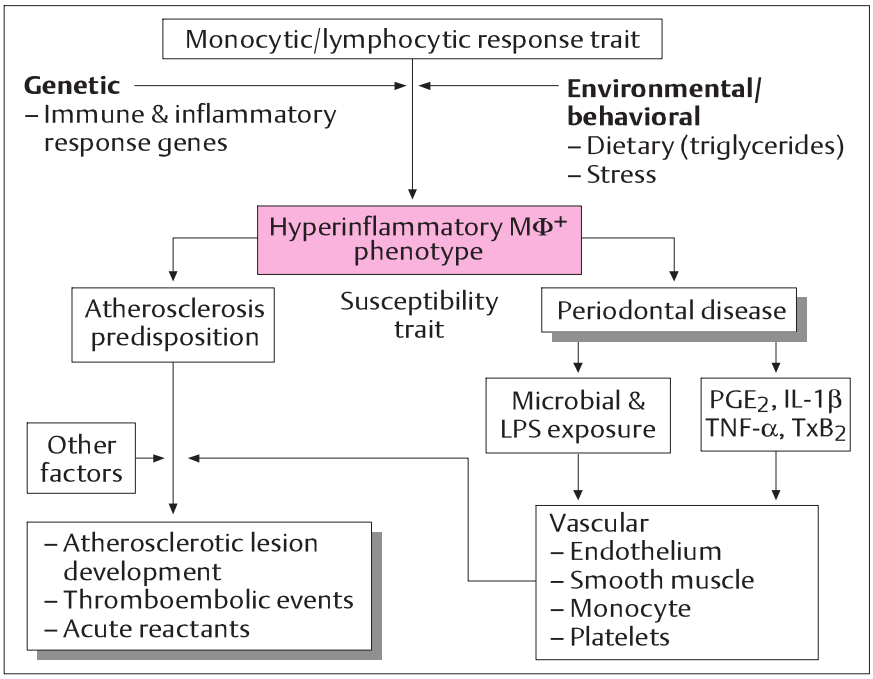


Fig. 8.1 The proposed model describing the possible link between vascular diseases and chronic periodontitis is based on the hypothesized existence of a so-called *hyperreactive macrophage phenotype*, which might be genetically controlled and modified by environmental and behavioral factors. In persons with increased susceptibility to the development of atherosclerotic vascular lesions, such lesions could develop after excessive release of proinflammatory mediators in the wake of chronic systemic exposure of the individual to bacteria and/or LPS during a chronic infection such as periodontitis. *MΦ* macrophage. (Adapted from Beck et al. 1996)

- Chronic infections increase the risk of atheroma development and thromboembolic events (Fig. 8.**1**). It is conceivable that atherosclerosis and periodontitis are linked to a special, *hyperresponsive monocyte/macrophage phenotype:*
  - Excessive release of proinflammatory cytokines and mediators (IL-1β, TNF-α, PGE_2) after contact with LPS of gram-negative bacteria.
  - Genetically controlled and modified by environmental and behavioral factors:
    - Genetically determined inflammatory and immunological host response.
    - Diet: low density lipoproteins (LDL), triglycerides; high-fat diet leads to increased secretion of proinflammatory and catabolic cytokines.
    - Stress.

Diabetes Mellitus

- Diabetes mellitus is a *heterogeneous group of diseases*. Common characteristics are:
  - impaired glucose tolerance;
  - altered lipid and carbohydrate metabolism.
- Two main forms of diabetes mellitus are differentiated:
  - *Type I:* cell-mediated autoimmune destruction of the insulin-producing β-cells of the pancreas. Genetic predisposition; onset may be triggered by virus infection:
    - High susceptibility to ketoacidosis.
    - Onset usually in children and adolescents.
    - Insulin substitution is required.
    - Patients are thin or have a normal stature.
  - *Type II:* alteration of insulin receptors with increase in insulin resistance of target tissues:
    - Genetic predisposition.
    - In most cases insulin production is adequate.
    - Resistance to ketoacidosis.
    - Onset usually in adults; however, incidence in adolescents is increasing.
    - Patients are frequently obese; characteristic facial redness (diabetic rubeosis).
    - Triglyceridemia.
    - Therapeutic control by (1) diet and weight reduction, (2) diet plus orally administered antidiabetic drugs, or (3) a combination of diet, oral antidiabetic drugs, and insulin.
- In addition, further *diagnoses* related to diabetes may be made:
  - Impaired glucose tolerance.
  - Impaired fasting glucose.
  - Gestational diabetes.
  - Diabetes due to diseases of the pancreas, certain drugs, endocrine disorders, infections, and genetic syndromes.
- The *prevalence* of diabetes mellitus in industrialized countries is about 8 %:
  - 85–90 % type II diabetes mellitus.
  - 5–10 % type I diabetes mellitus.
  - Prevalence increases with age and reaches 18–20 % above the age of 60 years.
  - About one-third of cases are undiagnosed.
- Classic symptoms, resulting from *hyperglycemia*, are:
  - Triad of polyuria, polydipsia, polyphagia.
  - In addition: itching, weakness, fatigue.
  - Increased susceptibility to infection.
- Hyperglycemia leads to:
  - Osmotic diuresis
  - Worsening of tissue oxygen supply and perfusion
  - Impaired chemotaxis, phagocytosis, and adhesion of neutrophilic granulocytes
  - Effect on *collagen metabolism:*
    - Synthesis, maturation, and homeostasis of collagen are strongly influenced by the glucose concentration in the tissue.
    - Collagenolytic activity is increased in diabetics.
- Other *pathogenetic mechanisms:*
  - Hyperglycemia leads to reversible glycation of lipids and proteins known as Amadori products.
  - If hyperglycemia is sustained, irreversible, nonenzymatic formation of advanced glycated end products (AGEs) occurs.
    - The prototype is glycated hemoglobin HbA_{1c}.
    - Marker of diabetic control.
  - AGEs alter the form and function of numerous extracellular matrix components, including collagen.
  - AGEs react with specific receptors (RAGEs) on diabetic target cells:
    - Endothelial and mesangial cells
    - Neuronal cells
    - Monocytes/macrophages

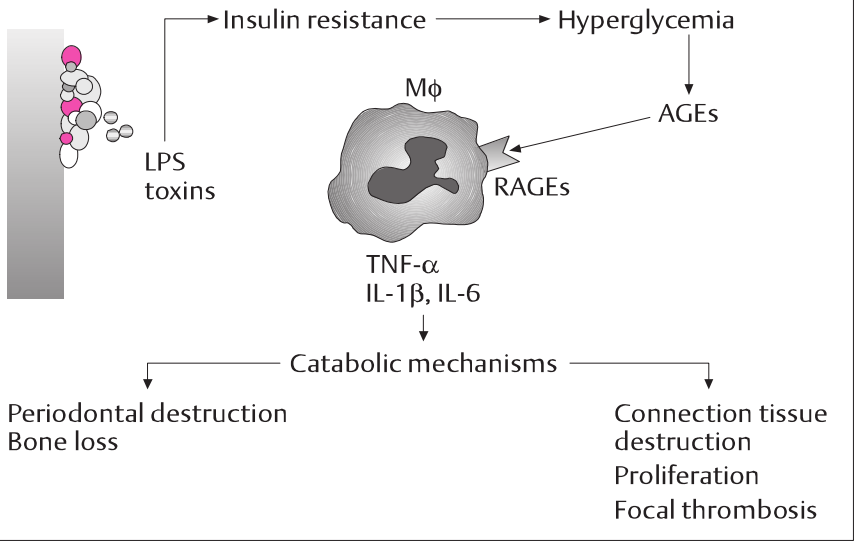


Fig. 8.**2** Possible mutual interrelationship between periodontitis and diabetes mellitus. Chronic infection with gram-negative bacteria in dental plaque leads, in diabetic patients, to increasing insulin resistance of the tissues and increasing hyperglycemia. This may result in an accumulation of irreversibly altered proteins (advanced glycated end products, AGEs), which bind to receptors on macrophages (RAGEs) and induce excessive release of proinflammatory cytokines, giving rise to a more catabolic situation

  - This leads to:
    - Vascular dysfunction, vasoconstriction, and focal thrombosis
    - Impaired wound healing
    - Excessive reactivity of monocytes releasing large amounts of proinflammatory mediators and insulin-like growth factor (IGF)
- ➤ *Note:* AGE-mediated processes play a central role in the pathogenesis of diabetic complications (Fig. 8.**2**):
  - Retinopathy
  - Nephropathy and, consequently, renal hypertension
  - Neuropathy
  - Macroangiopathy (atherosclerosis and, subsequently, cardiovascular, cerebrovascular, and peripheral vascular disease)
  - Impaired wound healing
  - Periodontal alterations—according to some authors the "sixth complication" of diabetes mellitus
- ➤ *Note:* Glycemic control is prevention of complications.
- ➤ Diabetes mellitus and *periodontitis:*
  - Periodontal alterations (Fig. 8.**3**) usually occur in patients with poorly controlled or undiagnosed diabetes mellitus:
    - Tendency to enlargement/proliferation of the gingiva
    - Increased bleeding tendency
    - Tendency to periodontal abscess formation
    - Increased progression rate of periodontal destruction
  - Overall, diabetics have a two to three times higher risk of periodontitis than nondiabetics.
  - Poorly controlled diabetics have a strongly increased risk of progressive periodontitis: relative risk is greater than 10.
  - Mutual interrelationship:
    - Chronic infections impair metabolic control: the insulin resistance of the tissues increases.

Fig. 8.**3** Clinical appearance of periodontal disease in a patient with type II diabetes mellitus. Hemorrhagic, thickened, and proliferative gingiva with a tendency to abscess formation

Table 8.**3** Diabetes diagnosis

Patient with suspected undiagnosed diabetes mellitus	Known diabetic patient
• Symptoms of diabetes mellitus – Polyuria – Polydipsia – Polyphagia – Unexplained loss of weight, and • Plasma glucose ≥ 200 mg/dl (not fasting) • Fasting blood glucose: – Diabetic patient: ≥ 126 mg/dl – Normal: 70 – 110 mg/dl – Impaired: 110 – 126 mg/dl • Impaired glucose tolerance: plasma glucose two hours after intake of an equivalent of 75 g anhydrous glucose dissolved in water – Diabetic patient: ≥ 200 mg/dl – Normal glucose tolerance: <140 mg/ml – Impaired tolerance: 140 – <200 mg/ml *Note:* As a rule, the findings should be confirmed on another day using one of these three methods	Glycated hemoglobin: reflects average blood glucose level of the last four to six weeks • Normal: HbA_1 5.5–8.4 % HbA_{1c} 4.4–6.7 % • HbA_{1c} <8 %: well controlled (aim: <7 %) • HbA_{1c} 8 – 10 %: moderately controlled • HbA_{1c} >10 %: poorly controlled

- Periodontal treatment has favorable effects on metabolic control. Adjunctive systemically administered doxycycline may be considered: it is not metabolized in the kidney (risk of nephropathy) and inhibits tissue collagenase.

➤ If diabetes mellitus is suspected, appropriate tests should be arranged immediately (Table 8.**3**). The use of a self-monitoring glucometer may be recommended even in the dental practice:
- Rapid and cost-effective
- To confirm an initial suspicion
- To check blood glucose level in known diabetics
- *Note:* Quality control is mandatory.

Pregnancy

➤ Severe periodontal disease of the expectant mother seems to increase the risk of preterm labor and *low birth weight* (below 2500 g):
- Case control studies have calculated an odds ratio of about 7 after adjusting for known risk factors.
- Preliminary interventional studies showed that the risk decreases after periodontal treatment.

➤ During pregnancy, various alterations of the periodontal condition may be observed which frequently bring patients to the periodontist:
- Marked pregnancy gingivitis
- Pyogenic granuloma (Fig. 8.**4**)
- Worsening of existing periodontitis
- Physiological increase in tooth mobility towards the end of the pregnancy.

➤ Periodontal infections should be checked over from the earliest stage of pregnancy, with:
- Treatment of open carious lesions.
- Establishment of effective oral hygiene.
- Subgingival scaling.
- Review of dietary behavior.
- Regular supportive care.

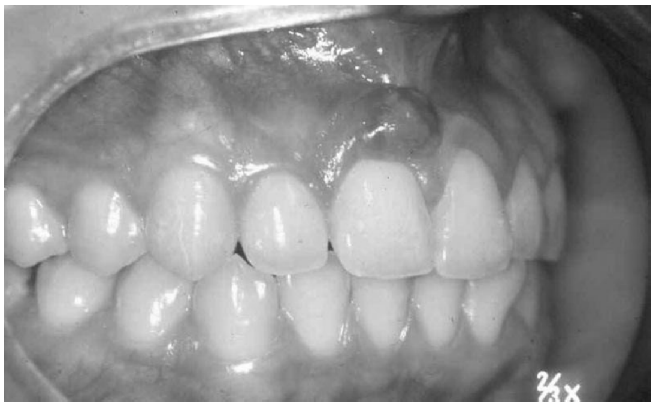


Fig. 8.**4** During pregnancy, a pyogenic granuloma may develop as a response to local irritation

- *Note:* There are no objections to intraoral adjunctive use of chlorhexidine-containing mouthrinses and gels during pregnancy.

➤ The aim is a reduction in the load of oral pathogens (*S. mutans*, *A. actinomycetemcomitans*, *P. gingivalis*) in the expectant mother
- to reduce the risk of early transmission to the child and thus
- inhibit the development of caries and inflammatory periodontal diseases.

Osteopenia, Osteoporosis

➤ Osteopenia and osteoporosis most often affect women after the menopause.
- High levels of estrogen before the menopause and estrogen replacement thereafter protect against osteoporosis and reduce the risk of periodontitis and tooth loss.
- If osteopenia or osteroporosis is suspected, the patient should be referred to her gynecologist for bone density measurement. Hormone replacement protocols include:
  - Estrogen and progesterone in women still having an uterus
  - Estrogen only in women after hysterectomy
  - If required, combined medication with NaF, calcium, vitamin D, or diphosphonate.
- In view of new evidence regarding considerable other risks, the benefits of hormone replacement therapy have been seriously questioned.

Fig. 8.**5** Typical clinical appearance of the dentition of a heavy smoker. Note melanosis of the gingiva, in particular in the mandible. Hyperkeratosis of the gingiva, recession, heavy extrinsic stain, and calculus formation, but no overt cardinal signs of gingival inflammation

Smoking

➤ Smoking is the most important avoidable risk factor for many serious and life-threatening diseases, e. g.:
- Chronic obstructive lung disease
- Lung, oral, and throat cancer
- Cardiovascular disease

➤ The prevalence of smoking is high in most European countries:
- In Germany, about 37 % of adult men and 22 % of adult women smoke.
- The highest rate is found among 25- to 40-year-olds: 48 % of men and 36 % of women.
- In Kuwait 52 % of adult men and 12 % of adult women smoke.

➤ Smokers are easily identified during dental examination (Fig. 8.**5**):
- A characteristic feature is *smoker's melanosis*, which affects about 20–30 % of cases, especially heavy smokers.
- Externally stained teeth and marked *calculus* formation.
- Only minor signs of gingival inflammation in the presence of plaque:
  - Less redness and edematous swelling
  - Reduced tendency to bleeding on probing

➤ *Note:* In order to detect early signs of cancer or precancerous conditions (leukoplakia, smoker's leukokeratosis) in heavy smokers, a careful inspection of the oral cavity is mandatory (cf. Fig. 7.**2**).

➤ Pathogenetic mechanisms:
- Tobacco smoke affects the first line of defense against infection:
  - Reduced chemotaxis and phagocytosis of neutrophilic granulocytes
  - Diminished antibody production.
- Monocytic release of proinflammatory mediators is stimulated.
- Increasing proportions of obligately anaerobic periodontal pathogens such as *T. forsythia* and *P. gingivalis* are present in subgingival plaque.
- At high doses, nicotine damages gingival and desmodontal fibroblasts.

➤ Epidemiological data confirm the role of smoking as the main risk factor for development and progression of periodontal disease:
- For periodontitis, a relative risk of cigarette smoking of 2.5–6 has been reported.

- In children and adolescents especially, the relative risk may be considerably higher (>10).
- Cigarette smoking is responsible for more than 50% of observed periodontitis in age groups up to 45 years.
- If smoking could totally be eliminated, the prevalence of periodontitis might be reduced by 30–60%.

➤ Impaired treatment results after periodontal therapy:
- Even in smokers, appropriate periodontal treatment leads to a reduction in probing depth.
- However, attachment gains may be 25–60% less than those achievable in nonsmokers. This holds true for all forms of treatment:
  - Nonsurgical therapy
  - Flap surgery
  - Regenerative measures
  - Plastic and periodontal surgery
  - Local antibiotic treatment
- The predictability of treatment outcome, especially after guided tissue regeneration, is considerably reduced in heavy smokers.

➤ Positive effects rapidly occur after smoking is stopped:
- Progression rate of periodontitis slows.
- Wound healing after periodontal surgery improves.

➤ *Note:* Contemporary dentistry with its special focus on prevention of oral diseases must include advice to the patient and, especially, medical support of the patient's attempts to definitively quit smoking:
- Regular assessment and documentation of smoking status; use of a special smoking questionnaire (Table 8.**4**)
- Medical counseling about smoking
- Ongoing nicotine replacement: nicotine patches, nicotine chewing gums:
  - *Note:* Less than 7% of individuals who attempt to quit smoking are abstinent after a year.
  - However, 25% of smokers who are abstinent after one week using nicotine replacement are still abstinent after a year.
- Cooperation with general practitioners, psychologists, etc. If required, antisympathotonic, antidepressive, or anxiolytic drugs may be prescribed.
- First aim should be to give up smoking at least during and for several weeks after periodontal surgery.

➤ In heavy smokers who are unwilling or unable to quit, early extraction of questionable teeth may be indicated.
- Patients should be informed about the considerably lower success rate after regenerative measures or implant placement.
- In any case, rigorous attempts have to be made to control periodontal infections.

Table 8.**4** Smoking questionnaire. (Adapted from Fowler 1997)

Are you now or have you ever been a smoker?	Yes No
At what age did you first smoke regularly (at least once a day)?	years of age
For how many years altogether have you smoked/did you smoke regularly?	years smoked regularly
Do you currently smoke at least once a day?	Yes No
On average, how many cigarettes do you smoke a day?	cigarettes per day
On average, how many cigars do you smoke a day?	cigars per day
On average, how much tobacco (pipe or roll-ups) do you smoke a day?	ounces of tobacco per week
In the last 12 months, have you seriously tried to give up smoking?	Yes No
In the last 12 months, has a doctor or nurse advised you to give up smoking?	Yes No
Do you think the amount you smoke is harmful to your health?	Yes No
Would you like to give up smoking?	Yes No Don't need to

Therapy

9 Emergency Treatment

Periodontal Emergencies

Traumatic Insults

- Improper use of oral hygiene remedies (brushing trauma, Fig. 9.**1**; clefts after misuse of dental floss) or thermal (e.g., hot cheese on pizza) or chemical insults may injure the gingiva.
- Differential diagnoses:
  - Necrotizing ulcerative gingivitis/periodontitis
  - Herpesvirus infections
- Treatment:
  - Suspend all mechanical oral hygiene measures
  - The injured area may be covered by periodontal pack (CoePak)
  - Mouthrinses twice daily with 0.1–0.2% chlorhexidine digluconate solution
  - Follow-up appointment after one week.

Necrotizing Ulcerative Periodontal Disease

- Various forms are distinguished depending on the extent of the disease and the topography of the lesions:
  - Necrotizing ulcerative gingivitis
  - Necrotizing ulcerative periodontitis (Fig. 9.**2**)
  - Necrotizing stomatitis
- Differential diagnoses:
  - Herpesvirus infection, especially herpetic gingivostomatitis
  - Traumatic injury
- Diagnosis and treatment:
  - Arrange differential blood count and HIV test to exclude:
    - Hematological diseases, especially agranulocytosis and leukemia
    - HIV infection
  - Local treatment:
    - Careful débridement.
    - Remove pseudomembranes covering necrotic areas with 3% H_2O_2.
    - Prescribe chlorhexidine preparations: 0.1–0.2% mouthrinse, 1% gel.
  - Follow up daily until complaints subside.
  - If complaints persist, and after any contraindications have been excluded, metronidazole (3 × 250 mg/day for seven days) may be administered systemically.
- *Note:* There is a high potential for regeneration of lost interdental papillae, especially in young persons with good oral hygiene:
  - Use of dental floss should be preferred to interdental brushes or tooth picks.
  - Gingivoplastic correction is only indicated if interdental craters persist.

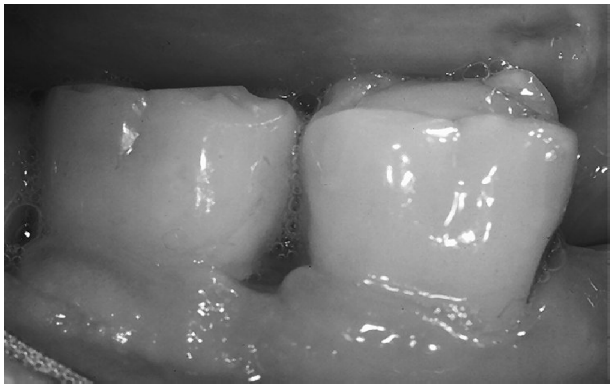


Fig. 9.**1** Loss of interdental papilla following improper use of interdental brush

Fig. 9.**2** Necrotizing ulcerative periodontitis in an HIV-seropositive patient

Fig. 9.**3** HSV-1 infection of gingiva. Shortly after vesicle development the vesicles have burst, leaving painful ulcerations. In this patient typical lesions were also seen on the upper lip

Herpetic Gingivostomatitis

- Herpetic gingivostomatitis is the primary infection with the herpes simplex virus (HSV-1):
  - Peak incidence between 2 and 4 years.
  - May occur in young adults: associated with HIV infection?
- Differential diagnoses:
  - Necrotizing ulcerative gingivitis
  - Erythema multiforme
- Treatment:
  - Superinfection with pathogenic bacteria must be avoided:
    - Local débridement
    - Prescription of chlorhexidine preparations
  - Virustatic medication may be given, e.g., aciclovir.
  - Depending on extent of the disease, patient may be referred to a pediatrician or physician
  - If appropriate, an HIV test should be arranged.
- Recurrent herpes lesions (cold sore, Fig. 9.**3**) do not usually require any special treatment.

Periodontal Abscess

- Differential diagnoses:
  - Acute apical periodontitis
  - Dentoalveolar abscess
  - Langerhans cell histiocytosis
- Diagnosis and treatment:
  - Vitality testing, periapical radiograph.
  - Local anesthesia.
  - Surgical draining; incision, if possible, from the entrance of the pocket (Fig. 9.**4**).
  - Careful débridement, instillation of 1% chlorhexidine gel.
  - Systemic antibiotics may be prescribed if required: azithromycin, or amoxicillin in combination with a β-lactamase inhibitor, e.g., clavulanic acid.

Fig. 9.**4** **a** Periodontal abscess at first premolar, which is vital.
b Incision from the entrance of the pocket.
c Situation after remission. In order to treat the lesion appropriately, periodontal surgery should now be carried out

- Multiple periodontal abscesses may develop after prescription of antibiotics not resistant to β-lactamase. If this occurs:
  - Stop antibiotic treatment.
  - Carry out microbiological diagnosis (culture, if possible with antibiotic susceptibility testing).
  - Follow up daily until complaints subside.
- Start systematic periodontal treatment.

Combined Periodontic–Endodontic Lesions

➤ Differential diagnosis and treatment:
- *Endodontic cause:*
  - Spread of a periapical infection along the periodontal ligament.
  - Tooth does not respond to vitality testing and is presumably nonvital.
  - Root canal treatment; if prognosis is unclear, carry out preparation in due order and apply a temporary dressing, e.g., calcium hydroxide
  - For the time being, no root surface instrumentation.
  - Radiographic follow-up after three and six months.
- *Periodontal cause:*
  - Retrograde pulpitis; sensitivity testing yields a vital tooth.
  - Decide whether to preserve the tooth. *Note:* Bone loss as far as the apex usually indicates a hopeless case.
  - If considered possible, root canal treatment; if prognosis is unclear, carry out preparation in due order and apply a temporary dressing with, e.g., calcium hydroxide.
  - Thorough root surface instrumentation.
  - Radiographic follow-up after three and six months.
- Frequently, the cause of a periodontic–endodontic lesion is not clear:
  - Therefore, treatment of both sources of infection is necessary, i.e., root canal débridement and periodontal treatment.
  - The value of attempting tooth preservation is usually questionable.

10 Phase I: Cause-Related Treatment

Mechanical Plaque Control

General Remarks

- The aim of cause-related treatment is to reduce significantly the number of oral pathogens present in the mouth. First the ecological conditions in the oral cavity have to be altered:
  - Hopeless teeth should be extracted, sometimes with incorporation of temporary dentures, and/or treatment to restore function may be given.
  - Open carious lesions should be treated and defective restorations (marginal gaps, overhanging fillings and crowns) corrected or exchanged.
  - Endodontic treatment may be carried out if required.
  - *Note:* The patient can only be motivated and instructed in proper oral hygiene measures if his or her ability to practice oral hygiene has been restored (see below).
  - Supra- and subgingival scaling, which should always be considered definitive, should be carried out.
- Effective oral hygiene may be particularly complicated in periodontally diseased patients, which requires a revision of the patient's ideas of the time it takes to properly clean the teeth: there may be
  - open interdental embrasures;
  - large, exposed root surfaces.
- Thorough cleaning should be performed once per day, e.g., before going to sleep, with:
  - Systematic toothbrushing.
  - Systematic cleaning of interdental tooth areas.
  - If possible, checking remaining plaque with a plaque disclosing agent, e.g., erythrocin, followed by its careful removal.
  - *Note:* All patients should additionally perform simple tooth brushing and mouth rinsing after every meal.
- Improvement of oral hygiene and reduction of bleeding tendency after probing should be recorded in the appropriate forms (cf. Fig. 7.**11**).

Toothbrushing Techniques

- Dental plaque is the cause of caries and inflammatory alterations of the periodontium:
  - Therefore, improvement of oral hygiene is mandatory at the start of every treatment.
  - Dental plaque is a biofilm, similar to that found on all surfaces of standing or flowing water and water supplies, sewerage, and sanitary installations.
  - In general, biofilms can only be removed mechanically.
- So far as changing the patient's behavior is concerned, a major aspect of communication is appropriate explanation of the problem—what one might call *motivating the patient:*
  - Hardly visible plaque should be revealed after disclosing:
    - In general, colored agents should be used, e.g., aqueous solutions of 3% erythrocin.
    - *Note:* Before using a plaque disclosing agent the mouth should be rinsed out with water to remove any viscous saliva from the tooth surfaces.
  - Problematic areas should be inspected with the patient (who holds a hand mirror): molars, interdental and cervical areas.
  - The close relationship between plaque and bleeding gums, and, if applicable, pockets, should be visualized.
- Numerous *toothbrushing techniques* have been developed for various purposes and situations:
  - The *scrub technique* (horizontal brushing) is preferred by most patients:
    - *Note:* Plaque removal with this technique is not very effective.
    - There may be a risk of injury.
  - *Modified Bass technique:*
    - A dry, multitufted brush with a short head is chosen.

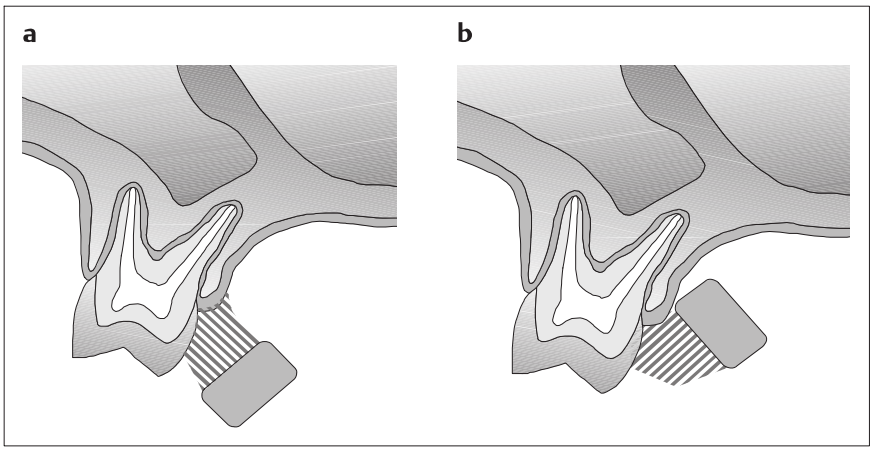


Fig. 10.**1** Toothbrushing techniques.
a Placement of the brush for modified Bass technique to remove gingival and sulcus plaque in particular.
b Charters technique to make a start on removing interdental plaque

- A small amount of toothpaste (pea size) is used.
- The head of the brush is placed on the tooth surface at an angle of 45° to the tooth axis, with the bristles directed to the gingival margin (Fig. 10.**1 a**).
- Small vibrations of the brush loosen cervical and sulcus plaque.
- The brush is then rotated coronally to wipe the plaque off the tooth surface.

– *Charters technique:*
- The brush is directed towards the crown of the tooth at an angle of 45° (the opposite way to the Bass technique).
- This technique is particularly suitable for patients with interdental embrasures (Fig. 10.**1 b**).

– *Modified Stillman technique:*
- The brush is directed at an angle of 45° towards the gingiva and rolled towards the crown (roll technique).
- This technique is particularly suitable for patients with gingival recessions.
- There is virtually no risk of injury.

➤ Rather than laying stress on learning a completely new and unaccustomed technique, the emphasis at first should be on becoming *methodical:*
– Brushing should start in the difficult-to-reach, habitually uncleaned areas.
– Cleaning moves from one tooth group to the next, e. g.:
- Start lingually at the left side of the lower jaw: wisdom tooth, molars, premolars, canine, incisors, right canine, premolars, molars, wisdom tooth.
- Go on to the palatal surfaces in the upper jaw: start on the right side in the wisdom tooth/molar region and go on to the left side.
- Change to the buccal surfaces in the upper jaw: start on the left side and go on to the right side.
- Go on to the buccal surfaces of the lower jaw: start on the right and continue on the left.
- Finally, brush the occlusal surfaces.

– Innovative brushes with oblique, longer bristles should make it possible to improve interdental cleaning.

➤ In general, *electric toothbrushes* make oral hygiene easier. Innovative concepts that have proved themselves particularly well are:
– oscillating and rotating brush heads;
– use of acoustic energy to
- loosen the biofilm;
- even kill fastidious periodontal pathogens (?).

➤ Irrigating devices cannot remove structured plaque from tooth surfaces. If recommended, they should be used only in combination with antimicrobial agents.

Interdental Hygiene

➤ *Note:* Interdental areas are usually not adequately reached by normal or electric tooth brushes:
– Therefore, additional *special aids* are required for interdental cleaning:
- Dental floss

Fig. 10.**2** Dental flossing technique. The floss is wound around the middle fingers and cautiously guided by thumbs and index fingers across the contact area with sawing movements. The proximal surface is cleaned by moving the floss up and down, keeping tight contact

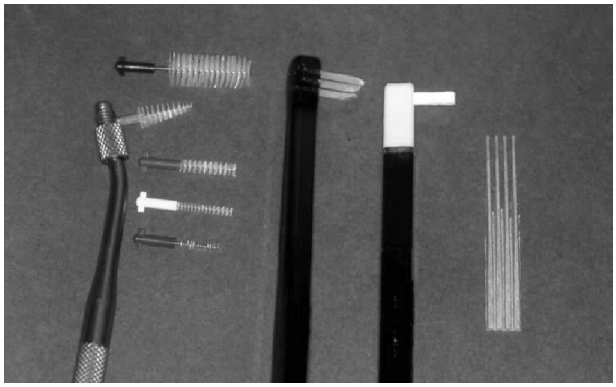


Fig. 10.**3** A large array of aids for interdental tooth cleaning is available. The presence of interdental embrasures of different sizes may cause problems, since more than two different interdental brushes are generally not accepted by the patient, and, furthermore, the risk of injuries increases

  - Interdental brushes
  - Tooth picks
- *Note:* Interdental cleaning can only be performed if the interdental embrasures are free and open (no calculus, no overhangs of fillings or crowns).

➤ *Dental floss* (Fig. 10.**2**):
- Available waxed or unwaxed; has the broadest range of applications:
  - About 40 cm of dental floss is wound around the middle fingers of both hands.
  - With thumbs and index fingers the floss is then cautiously guided across the contact area of the teeth.
  - The floss is moved up and down in contact with the proximal tooth surface to be cleaned.
  - *Note:* after five to six movement cycles the floss will make a squeaking sound—a sign that the tooth surface is clean (waxed floss and Teflon floss will not squeak).
- Special dental floss (superfloss) consisting of three parts is available for pontics and soldered crowns:
  - Stiff end to thread
  - Spongy intermediate part to clean the gingival side of the pontic
  - Longer part made of normal dental floss
- Disadvantages of flossing:
  - Demanding technique
  - Risk of injury
- Electrically driven dental flosses are not recommended.

➤ *Interdental brushes* (Fig. 10.**3**):
- In patients with wide open interdental embrasures and concave root areas:
  - Since interdental embrasures usually vary in size in any patient, more than one interdental brush may be needed.
  - After careful analysis of existing conditions (passage, friction), tailored advice should be given.
  - *Note:* Never recommend more than two interdental brushes. Having too many aids is always counterproductive.
  - For large embrasures, interdental brushes without a handle should be prescribed.
- If different interdental aids are necessary for different embrasures, special, individually completed forms may be useful for the patient's homecare.

Chemical Plaque Control

General Remarks

➤ Antimicrobial agents may be incorporated into oral health remedies to support the effects of mechanical plaque control:
- Toothpastes and gels
- Rinsing solutions

- Toothpastes are particularly suitable:
  - Most people brush their teeth at least once a day.
  - The importance of fluorides as an active compound in toothpastes for caries prevention has been well known for many years.
  - *Note:* The wide distribution of fluoride-containing toothpastes is the most important reason for the observed decline in caries in most western industrialized nations.
- The rather complicated *composition of toothpastes* may interfere with antibacterial additives. In general, toothpastes contain the following constituents:
  - *Abrasive components:* calcium carbonate and phosphate, metaphosphate, silica, aluminum oxide
  - *Suspension media:* water, glycerin, propylene glycol, sorbitol syrup
  - *Thickening, stabilization, and binding agents:* gels, starch, alginate, oily substances
  - *Detergents* such as sodium lauryl phosphate
  - *Aromatic compounds:* essential oils such as menthol, or peppermint oil, to improve taste; they may also exert antibacterial effects.
  - *Medical and chemical ingredients:*
    - Fluorides
    - Antimicrobial agents such as, e.g., metal salts, triclosan
    - Enzymes, e.g., amyl glucosidase, glucose oxidase
    - Antiinflammatory compounds
    - Tartar (calculus)-inhibiting substances such as diphosphonate, pyrophosphate, triclosan
    - Desensitizing substances such as potassium and strontium salts, hydroxyapatite
    - Vitamin A
    - Dyes
- There is usually a limit to what can be achieved by purely mechanical plaque control. Breakthroughs in individual oral hygiene are made by employing different kinds of chemical plaque control: "*soft chemoprophylaxis*" (Table 10.**1**).

Table 10.**1** Some antimicrobial compounds and related products to reduce plaque and gingivitis

Compounds	Examples	Mechanism of action	Products
Bisbiguanide	Chlorhexidine	Antimicrobial	Toothpastes, gels, mouthwash, spray, chewing gum, varnishes
Quaternary ammonium compounds	Cetylpyridinium chloride, benzalconium chloride	Antimicrobial	Mouthwash
Phenolic compounds, essential oils	• Thymol, menthol, eucalyptol • Triclosan	• Antimicrobial • Antimicrobial, anti-inflammatory	Mouthwash, toothpastes
Metal salts	• Tin, zinc • Strontium, potassium	• Antimicrobial • Desensitizing	Mouthwash, toothpastes
Fluorides	Sodium fluoride, sodium monofluorophosphate, stannous fluoride, amine fluoride	Caries-inhibiting, antimicrobial, desensitizing	Toothpastes, gels, mouthwash, varnishes
Amino alcohols	Delmopinol	Interferes with biofilm formation	No product available
Oxygen-releasing compounds	Hydrogen peroxide, sodium perborate, sodium percarbonate	Antimicrobial	Mouthwash
Plant products	Sanguinarine	Antimicrobial	Mouthwash, toothpastes
Enzymes	Glucose oxidase, amyloglucosidase	Antimicrobial	Toothpaste

➤ The following factors should be considered:
- *Substantivity:* the substance should adhere to surfaces of the oral cavity as strongly as possible, so that it is *released slowly* at concentrations that interfere with de novo plaque formation.
- *No adverse interaction* with other components of the toothpaste. *Note:* Despite excellent retention and subsequent sustained release, the plaque-inhibiting effect of cationic chlorhexidine in customary toothpaste formulations is neutralized by anionic detergents and calcium ions.

Chlorhexidine

➤ 1,1'-Hexamethylene-bis[5-(P-chlorphenyl)-biguanide]. As digluconate, acetate, or (less water-soluble) hydrochloride; is the most efficacious oral antiseptic agent.
- Chlorhexidine has been firmly established in dentistry for several decades.
- In clinical studies for the development of new mouthwash formulations, it is usually the positive control.
- It has antimicrobial action against a broad spectrum of microorganisms:
  - Gram-positive and gram-negative bacteria
  - Fungi and yeasts, including *Candida* spp.
  - Some viruses (hepatitis B virus, HIV)
- Mouthrinse solutions usually contain 0.1–0.2 % chlorhexidine digluconate. A gel containing 1 % chlorhexidine but no detergents and abrasives and a spray containing 0.1 % chlorhexidine for disinfection of the tonsillar region are also available.
- Indications include:
  - Painful infections of the oral cavity if effective mechanical oral hygiene is not possible.
  - Postoperative infection control after periodontal or oral surgery.
  - Mentally retarded and/or physically handicapped patients; hospitalized patients.
- Preparations should be used only for a few weeks, since comparatively mild adverse effects may occur:
  - Discoloration of teeth and restorations
  - Hairy tongue
  - Taste alterations
  - Occasionally, desquamation of the epithelium
  - Rarely, swelling of the parotid gland

Triclosan

➤ 5-Chloro-2-(2,4-dichlorphenoxy)-phenol. Nonionic, lipid-soluble, antimicrobial substance with a broad range of action; does not interfere with detergents and other components of toothpastes. It is added to cosmetics and soaps as a preservative.
- Since substantivity is low, various strategies have been employed to increase oral retention:
  - Lipid-soluble triclosan is incorporated into a copolymer of polyvinylmethylether and maleic acid (PVM/MA, Gantrez), which has considerable retention capacity on oral surfaces
  - Triclosan is dissolved in polydimethylsiloxane (silicon oil)
  - Triclosan is combined with zinc citrate (additive plaque-inhibiting effect)
- An observed antiinflammatory effect appears to be independent of its antimicrobial effect:
  - Triclosan interferes with arachidonic acid metabolism.
  - After topical application, production of proinflammatory mediators PGE_2 and leukotriene B_4 is reduced.
- Use in toothpastes may even influence the composition of subgingival plaque and retard periodontitis progression.

Metal Salts

➤ Stannous fluoride, zinc citrate, and other metal salts have low toxicity but do have plaque-inhibiting effects; they may be used as components of toothpastes and mouthwashes:
- Metal ions interfere with bacterial metabolism; SnF_2 inhibits bacterial glycolysis.
- Zinc, tin, and copper ions eliminate volatile sulfur compounds of gram-negative bacteria (H_2S, CH_3SH), which are responsible for halitosis.

Other Additives

- Essential oils, phenolic compounds, plant extracts:
  - Some *essential oils* have a certain antimicrobial effect in vitro. There are contradictory results in relation to plaque inhibition.
  - Sanguinarine, an alkaloid of the Canadian bloodroot, has been incorporated into toothpastes and mouthwashes. Only minor plaque inhibition was observed.
- Agents influencing *calculus formation*:
  - Pyrophosphate
  - Triclosan in combination with Gantrez or zinc citrate
  - Diphosphonate
- Agents for treatment of *dentin hypersensitivity*:
  - There are different hypotheses about the mode of action of desensitizing ingredients:
    - They occlude dentinal tubules.
    - They cause coagulation or precipitation of tubular fluids.
    - They stimulate secondary dentin formation.
    - They block pulpal neural response.
  - *Note:* High concentrations of fluoride lead to precipitation of calcium fluoride and occlusion of dentinal tubules. This is the safest treatment for dentin hypersensitivity.
  - Metal salts (strontium chloride, potassium salts, sodium citrate) or formaldehyde have been incorporated into special toothpastes for the treatment of hypersensitive teeth, with varying results. Limited effects may be expected for potassium salts (nitrate or citrate), which may be superior to fluoride application.
- Further developments in the oral hygiene sector of health care require frequent revisions of current concepts:
  - Differing problems in different patients require individual solutions.
  - *Note:* The patient's oral hygiene in particular offers multiple opportunities for intensive communication.

Local Anesthesia

General Remarks

- The most important part of the first stage of periodontal therapy is supra- and subgingival scaling, which should be definitive. *Note:* The latter usually requires local anesthesia.
- Definition and mode of action:
  - Local anesthesia involves the regionally limited, reversible blocking of pain receptors or their afferent nerves.
  - The mode of action of local anesthetics is based on blocking sodium ion influx during afferent nerve conduction.
- Local anesthetics are quite toxic for the organism, so resorption should be kept to the minimum:
  - Vasoconstrictors prolong resorption time and reduce toxicity.
  - Surgical procedures may be carried out more easily under conditions of local ischemia.
- Local anesthetic agents are either amino esters of *p*-aminobenzoic acid or aminobenzoic amides. The respective residues of the molecule determine pharmacological properties and metabolism in the tissue:
  - Esters of benzoic acid: procaine, tetracaine.
  - Amino amides: lidocaine, prilocaine, butanilicaine, mepivacaine, articaine, bupivacaine.
  - Procaine, tetracaine, and to some extent also articaine may be hydrolyzed in the tissue: after some time, further injections exceeding the threshold dose may be possible.
  - All other anesthetics mentioned are either secreted unchanged or metabolized in the liver. *Note:* The threshold dose must not be exceeded in these cases.

Adverse Effects of Local Anesthetics

- *Intoxications* affect the central nervous system and the cardiovascular system:
  - Initial excitation phase: restlessness, tremor, anxiety, nausea, vomiting, visual deficits, tachycardia, rise in blood pressure.
  - Later: loss of orientation, tonic spasms, dyspnea.
  - If not treated immediately: loss of consciousness, drop in blood pressure, brady-

cardia; finally, circulation arrest and asphyxia.
- Under unfavorable circumstances (high endogenous release and exogenous supply of catecholamines, or accidental intravascular injection), *adrenaline intoxication* is possible despite careful consideration of any contraindications:
  - Increase in blood pressure
  - Tachycardia
  - Extra systoles, to the point of ventricular fibrillation

Regional Infiltration and Block Anesthesia

- An adequate depth of anesthesia is essential for subgingival scaling and periodontal surgery:
  - Root instrumentation usually leads to unpleasant dentinal pain.
  - Ischemia facilitates visualization of the surgical site.
  - Therefore, small amounts of quite highly concentrated anesthetics (2–4%) containing adrenergic vasoconstrictors (1:100 000) are injected.
- Procedure:
  - Most suitable are carpule systems equipped with a fine (ø 0.4 mm for block anesthesia) or extra-fine needle (ø 0.3 mm for infiltration).
  - In the mandible: block of the inferior alveolar nerve and the buccal, lingual, and mental nerves.
  - In the maxilla: infiltration anesthesia of the dental plexus together with a block of the major palatal nerve and the incisal nerve.
  - *Note:* The following techniques *should be avoided* because of the danger of traumatizing the gingiva and, especially, the periodontal ligament, as well as the likelihood of deep implantation of plaque bacteria:
    - Intrapapillary injections of small amounts to the base of the bony pocket.
    - So-called intraligamentary anesthesia; in fact, the anesthetic solution is spread via spongy bone.

Supragingival and Subgingival Scaling and Root Planing, Subgingival Curettage

General Remarks

- Definitive supra- and subgingival scaling and root planing is the most decisive procedure for the control of periodontal infections. Supragingival and subgingival scaling should be carried out in the same session.
- Working more or less only by tactile sensation requires considerable experience in assessing tooth and, especially, pocket morphology and in using the instruments.

Definitions

- *Scaling:* mechanical removal of plaque, calculus, and stain from coronal parts of the tooth and root surface.
- *Root planing:* removal of bacterially or toxically contaminated root cementum or dentin and leveling irregularities of the surface.
- *Curettage:* removal of pocket epithelium and gingival granulation tissue, usually using curettes.

Aims

- Maximum germ reduction of the oral cavity. Control of pocket infection by
  - removal of any endotoxin and bacterial deposits from the root surface;
  - removal of bacterially infiltrated root cementum;
  - if necessary, removal of pocket epithelium.
- To achieve an optimum healing result after creation of a biocompatible root surface.

Indication

- Periodontal pockets with a depth of 3 mm or more.

Contraindication

- Shallow pockets up to 3 mm.

Table 10.**2** Standard instruments of tray for mechanical débridement of teeth

Instruments	Description	Article no., manufacturer
Mouth mirror	Plane, front surface rhodium-coated, ∅ 22 mm	M4C, Hu-Friedy
Dressing pliers		DP18 or DP17, Hu-Friedy
Explorer	For subgingival calculus detection	EXD5, Hu-Friedy
Periodontal probe	Calibration in 1-mm steps or color coded 3–3–2–3 mm	PCPUNC15 or PCP11, Hu-Friedy
Scalers	Sickle scaler for • Anterior teeth and premolars: H6/H7 • Molars: CI2/3 or T2/3	 • SH6/76, Hu-Friedy • SCI2/36 or ST2/36, Hu-Friedy
Curettes	Universal curettes • Posterior teeth in the lower jaw: Langer 1/2 • Posterior teeth in the upper jaw: Langer 3/4 • Anterior teeth: Langer 5/6 Alternatively, area-specific curettes • Anterior teeth: Gracey 1/2 or 5/6 • Buccal/lingual surfaces of posterior teeth: Gracey 7/8 • Mesial surfaces of posterior teeth: Gracey 11/12 or 15/16 • Distal surfaces of posterior teeth: Gracey 13/14 or 17/18	 • SL1/26 or SL12AF, Hu-Friedy • SL3/46 or SL3/4AF, Hu-Friedy • SL5/66 or SL5/6MF, Hu-Friedy • SG1/26 or SAS1/2, or SG5/66 or SAS5/6, Hu-Friedy • SG7/86 or SAS7/8, Hu-Friedy • SG11/126 or SRPG11/126, or SRG15/16, Hu-Friedy • SG13/146 or SRPG13/146, or SG17/186, Hu-Friedy
Sharpening stone	Arkansas stone	SS4, SS299, Hu-Friedy

Instruments

➤ The following instruments are used for mechanical debridement (Table 10.**2**):
- *Scalers:*
  - H6/H7: straight, sickle-shaped scaler for the anterior and premolar region
  - CI2/3: angled scaler for the molar region
  - Taylor 2/3: as CI2/3 but smaller
- *Universal curettes* (Fig. 10.**4 a**): the face of the blade is honed at a 90° angle to the terminal shank. Both cutting edges can be used for all surfaces of each tooth, e.g.:
  - Barnhart 1/2
  - Columbia 4R/4L
- *Intermediates:* Langer curettes combine the shank design of Gracey curettes with a universal blade honed at 90°. Cutting edges of both sides of the blade. 1/2 less angled, adapts to mesial and distal surfaces of posterior teeth in the mandible; 3/4 sharply angled, for posterior teeth in the maxilla; 5/6 straight, for anterior teeth.
- *Area-specific curettes* (Fig. 10.**4 b**): the face of the blade is honed at about 70° to the terminal shank; one cutting edge only:
  - Gracey curettes: 1/2 or 5/6 for anterior teeth; 7/8 for buccal and lingual surfaces of posterior teeth; 11/12 (or 15/16) for mesial surfaces of posterior teeth; 13/14 (or 17/18) for distal surfaces of posterior teeth.

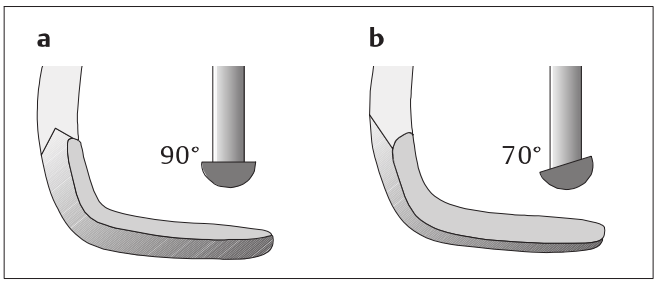


Fig. 10.**4**
a In universal curettes the face of the blade is honed at a 90° angle to the terminal shank, and there are two cutting edges.
b In area-specific curettes, the angle is 70°, and there is only one cutting edge

  - Gracey curettes are available with flexible terminal shanks for finishing, or a more rigid shank for removal of larger amounts of calculus. Tactile sensation is usually better with flexible curettes.
  - Both Gracey and Langer curettes are available with terminal shanks extended by 3 mm ("after-five") for deep pockets, and considerably shortened blades ("mini-five") for more slender incisor roots and narrow pockets (Fig. 10.**5**).
- *Periodontal files* are of only historical importance. They may be of use in exceptional cases, e.g., for narrow furcation entrances:
  - Orban files (relatively large)
  - Hirschfeld files (smaller)
- *Rotating instruments:*
  - Diamond-coated burs (75 or 40 µm) for smoothening the root surface after odontoplasty.
  - Extra-fine diamonds (15 µm; Perio-Set) for root planing.

➤ *Ultrasonic instruments* (magnetostrictive, piezoelectric; Table 10.**3**) make the removal of subgingival deposits considerably easier:
- Clinical results show they are as effective as hand instruments.
- Furcation areas in particular can be more effectively débrided.
- Innovative slim, microultrasonic tips allow instrumentation of deep, subgingival areas with sufficient water cooling.
- The cavitation effect of the water spray has a plaque-removing action.
- Acoustic energy may destroy sensitive bacteria (?).
- Antimicrobial agents may be used for rinsing.
- *Warning:* Damage to the root surface depends on tip angle and, especially, lateral force. Important points are:
  - A very shallow tip angle (tip should work parallel to the root surface).
  - Virtually no active force (≈ 0.5 N).
  - Especially for piezoelectric devices, the power setting adversely influences hard tissue removal.

➤ Similar guidelines apply for sonic scalers.

➤ Any surface roughness after scaling should be leveled by careful *polishing*:
- Nylon brushes or silicon cups with appropriate polishing paste should be used.
- Interdental spaces should be polished with special plastic spatulas for the Profine handpiece.
- Finally, fluoride gel or solution should be applied locally.

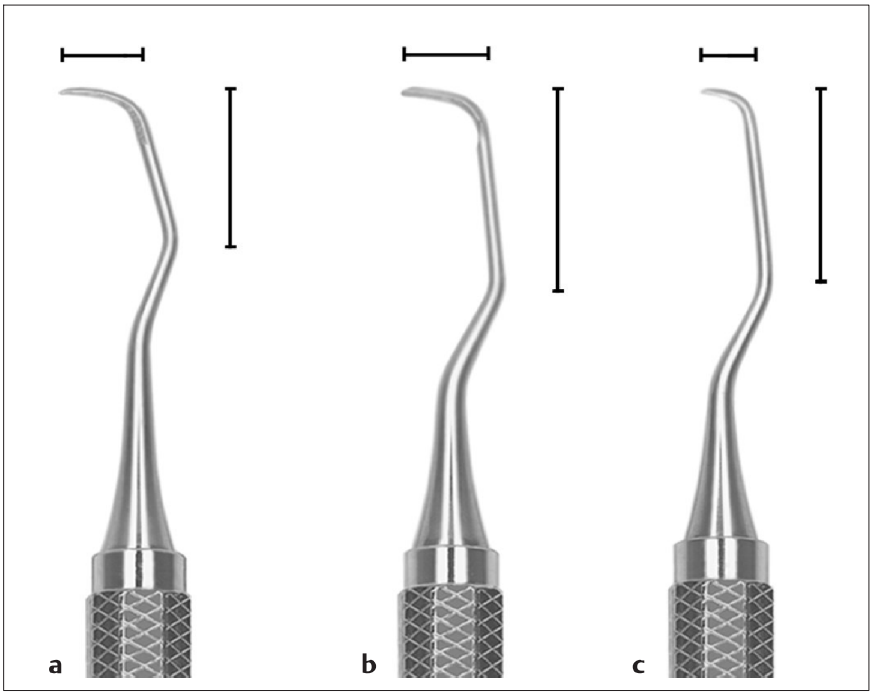


Fig. 10.**5** Area-specific curettes.
a Normal Gracey curette.
b After-five curette with a terminal shank extended by 3 mm.
c Mini-five curette with extended shank and blade shortened by about 50 %

Table 10.**3** Advantages and disadvantages of hand and ultrasonic instruments for root débridement

	Advantages	Disadvantages
Hand instruments	• Superior tactile sensation • Good access to tight pockets, particularly with miniaturized blades • Good adaptation to different root morphologies • No aerosol • No heat development	• Angulation of the blade of, e. g., 80° to root surface mandatory • Frequent sharpening required • Considerable working force for calculus removal • Tiring for the operator • Negative time factor
Ultrasonic instruments	• Current tips are very slender • Instrumentation virtually without pressure • Most surfaces can be reached, especially furcations • Destruction of the biofilm by cavitation • Bactericidal effect of acoustic energy (?) • Little soft tissue damage • Pocket irrigation with antimicrobial agents • Requires less time • No sharpening of tips • Better patient acceptance • Less tiring for the operator	• Poorer tactile sensation • Produces microscopic rippling of the root surface • Aerosol is highly contaminated • Not all handpieces can be autoclaved • Possible risk for patients with pacemakers • Contraindication in infectious patients

➤ Because of the aerosol created, special precautions for *cross-infection control* are necessary:
- For instance, two minutes' mouth rinsing with povidone-iodine (Betadine) or 0.1–0.2 % chlorhexidine solution for germ reduction before any treatment.
- Face mask and face shield should be worn.
- Suction function must be adequate.
- *Note:* Ultrasonic instrumentation should not be used in infectious patients (HIV, hepatitis) and patients with a pacemaker.

➤ Some oscillating instruments (e.g., Peri-o-tor) driven in a Profine hand piece will remove plaque without damaging the root surface.

Procedure

➤ *Subgingival curettage* consists of the following treatment steps:
- *Disinfection:* mouth rinsing with povidone-iodine or chlorhexidine solution for germ reduction.
- *Local anesthesia.*
- *Scaling:* removal of any soft and hard deposits from the root surface.
- *Root planing:* smoothening of the root surface and leveling of resorption lacunae in cementum which might be colonized by bacteria.
- *Soft tissue curettage:* removal of pocket epithelium. *Note:* Intentional removal of pocket epithelium is not regularly performed.

➤ Effective hand instrumentation depends on four critical factors:
- Adequate access to the bottom of the pocket
- Good fit between the curette and the root morphology
- Correct blade angle
- Thoroughness, i. e., complete root coverage

➤ *Note:* An intraoral fulcrum close to the working area, as traditionally recommended, may actually prevent effective blade angulation of 70–80°:
- Traditional techniques may prevent adequate access to the bottom of the pocket.
- More flexibility of the operator, alternative fulcrums, and alternative operator positioning are necessary:
  - In posterior areas of the maxilla, use of the mandible as an extraoral fulcrum is recommended (Fig. 10.**6 a**).
  - Posterior areas of the right mandible are operated on from a one o'clock position using the posterior teeth of the maxilla as fulcrum (Fig. 10.**6 b**).

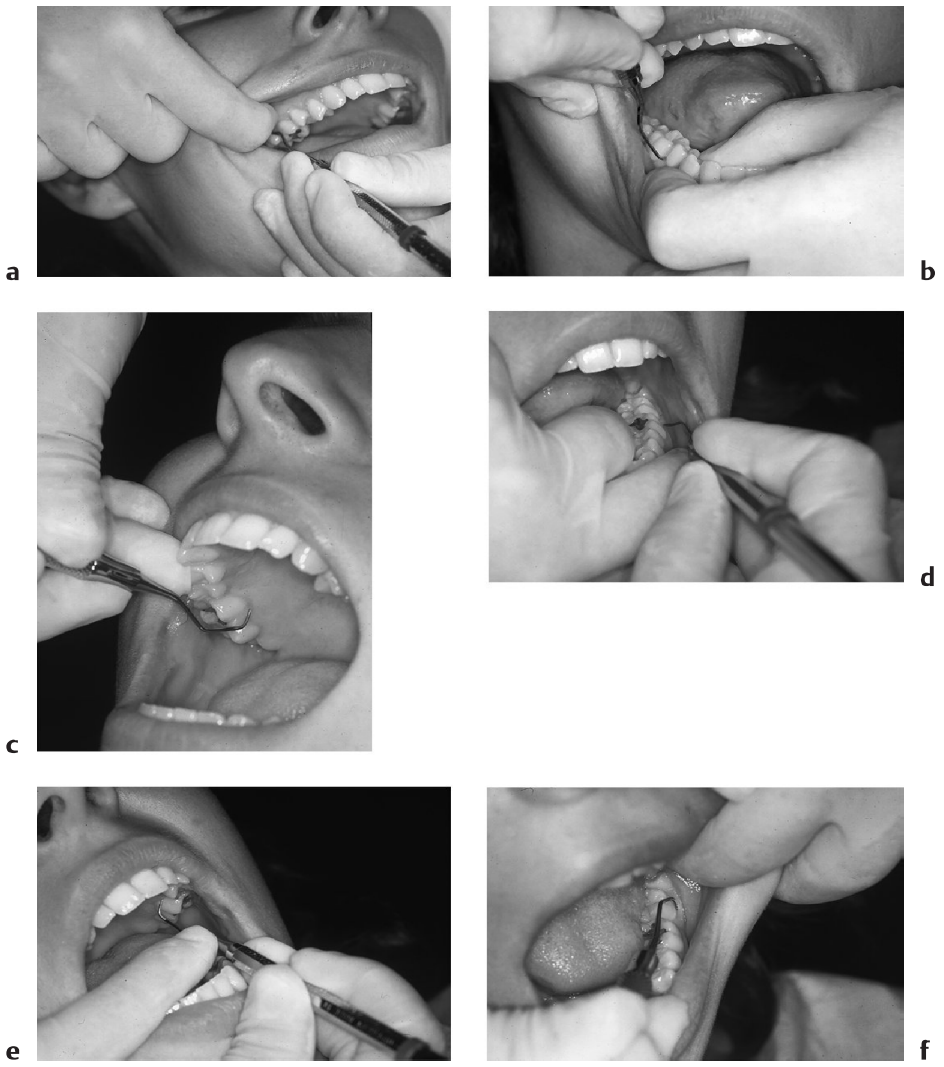


Fig. 10.**6** Subgingival curettage.
a Mandible as extraoral fulcrum during instrumentation of posterior areas of the maxilla.
b Maxillary teeth as fulcrum during instrumentation of posterior teeth of the mandible. The nondominant hand stabilizes the mandible.
c Instrumentation of posterior areas of the right maxilla from a two or three o'clock position.
d Fingers of the nondominant hand used as fulcrum.
e Reinforcement of the curette with the index finger of the nondominant hand.
f Horizontal stroke

- Posterior areas of the right maxilla are instrumented from a two or three o'clock position. The patient's head is tilted backwards (Fig. 10.**6 c**).
- The fingers of the nondominant hand may serve as a fulcrum (Fig. 10.**6 d**).
- In certain cases, an extraoral, reinforced fulcrum may be necessary. The curette is activated with the index finger of the nondominant hand (Fig. 10.**6 e**).

– The instrument is held in a modified pen grasp.
– The blade of the curette is inserted into the pocket, toe down, with an exploratory stroke.
– The working stroke is conducted with the blade correctly angled at 70–80°.
– In deep and narrow bony pockets, horizontal strokes are performed across the instrumented root surface, beginning at the bone level and proceeding coronally (Fig. 10.**6 f**).

➤ After removal of mineralized and nonmineralized deposits (*scaling*), *root planing* is carried out with universal and area-specific curettes:
– Bacteria generally colonize resorption lacunae in pathologically altered root cementum; these can only be leveled by thorough instrumentation.
– However, complete removal of root cementum is not desired:
  - Opening dentinal tubules increases the risk of bacterial penetration.
  - Risk of dentin hypersensitivity is increased
  - Reattachment is prevented.

- Instrumentation is carried out in an overlapping pattern.
- In practice, no distinction is made between scaling and root planing.

➤ *Soft tissue curettage*, e. g., in cases of slightly hypertrophic gingiva:
- A universal curette is inserted inversely into the pocket.
- The inner surface of the pocket is carefully peeled.
- *Note:* No evidence can be found in the literature that generally performed gingival curettage has any therapeutic benefit over scaling and root planing alone.

➤ The smoothness of the root surfaces is checked with an explorer.

➤ Rinsing with physiological saline, compression of widened gingival margins. In some cases periodontal pack (CoePak, PeriPak) should be applied for a couple of days.

➤ *Note:* In general, subgingival scaling should be carried out with both ultrasonic and hand instruments.

Critical Assessment

➤ The operator needs to get a feeling for the effectiveness of the procedure:
- *Efficacy* measures the extent to which an intervention does more good than harm under ideal conditions.
- *Effectiveness* measures the same under usual general practice circumstances.
- *Efficiency* measures the effect of the intervention in relation to the resources it consumes.

➤ In general, the clinical periodontal situation should improve considerably (Fig. 10.**7**):
- A reduced number of gingival units that bleed on probing.
- Reduced periodontal probing depths:
  - Pockets with a probing depth of ≥6 mm respond with a combination of gain in clinical attachment and gingival recession; as a rule of thumb, the deeper the pocket, the more gain in clinical attachment can be expected.
  - *Note:* Subgingival scaling in shallow pockets results in undesired gingival recession and attachment loss.
- The number of pockets for which periodontal surgery (e. g., flap surgery) is indicated should be greatly reduced.

➤ As to the subgingival microflora, some striking alterations have been observed after supra- and subgingival scaling and root planing (Fig. 10.**8**):
- Increase of periodontally inert *Actinomyces* spp. (which have been shown to be antagonists of some periodontal pathogens)
- Reduction of spirochetes and motile rods
- Reduction of established periodontal pathogens *T. forsythia*, *P. gingivalis*, and *T. denticola*.
- Most interestingly, the prevalence of other bacteria remains more or less unchanged.

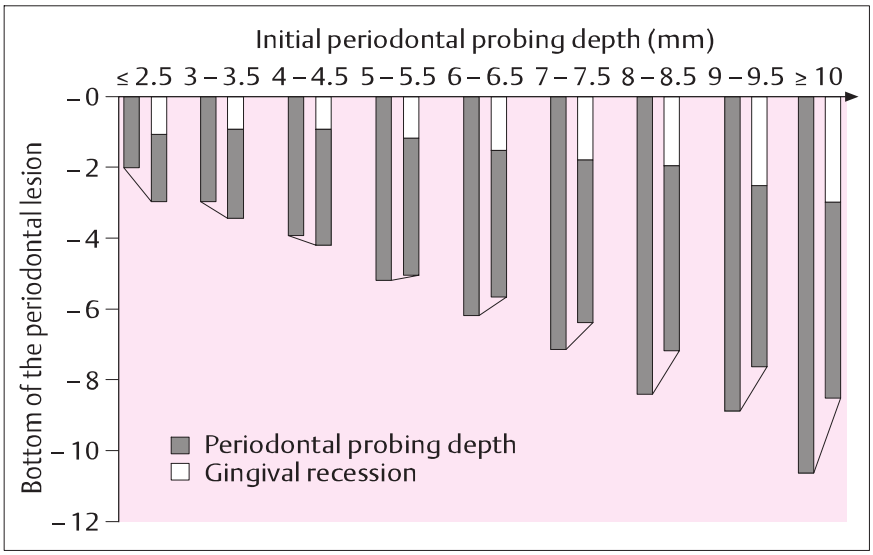


Fig. 10.**7** Average changes of probing parameters (periodontal probing depth, clinical attachment level, gingival recession) two years after subgingival scaling in relation to initial periodontal probing depth (Badersten et al. 1984). Sixteen adults with severe periodontitis were treated. Scaling in shallow pockets up to 4 mm led to considerable attachment loss. In deep pockets (6 mm or more), attachment gain was observed—the deeper the pockets, the greater the gain, e. g., about 2 mm in 10-mm-deep pockets, on average.

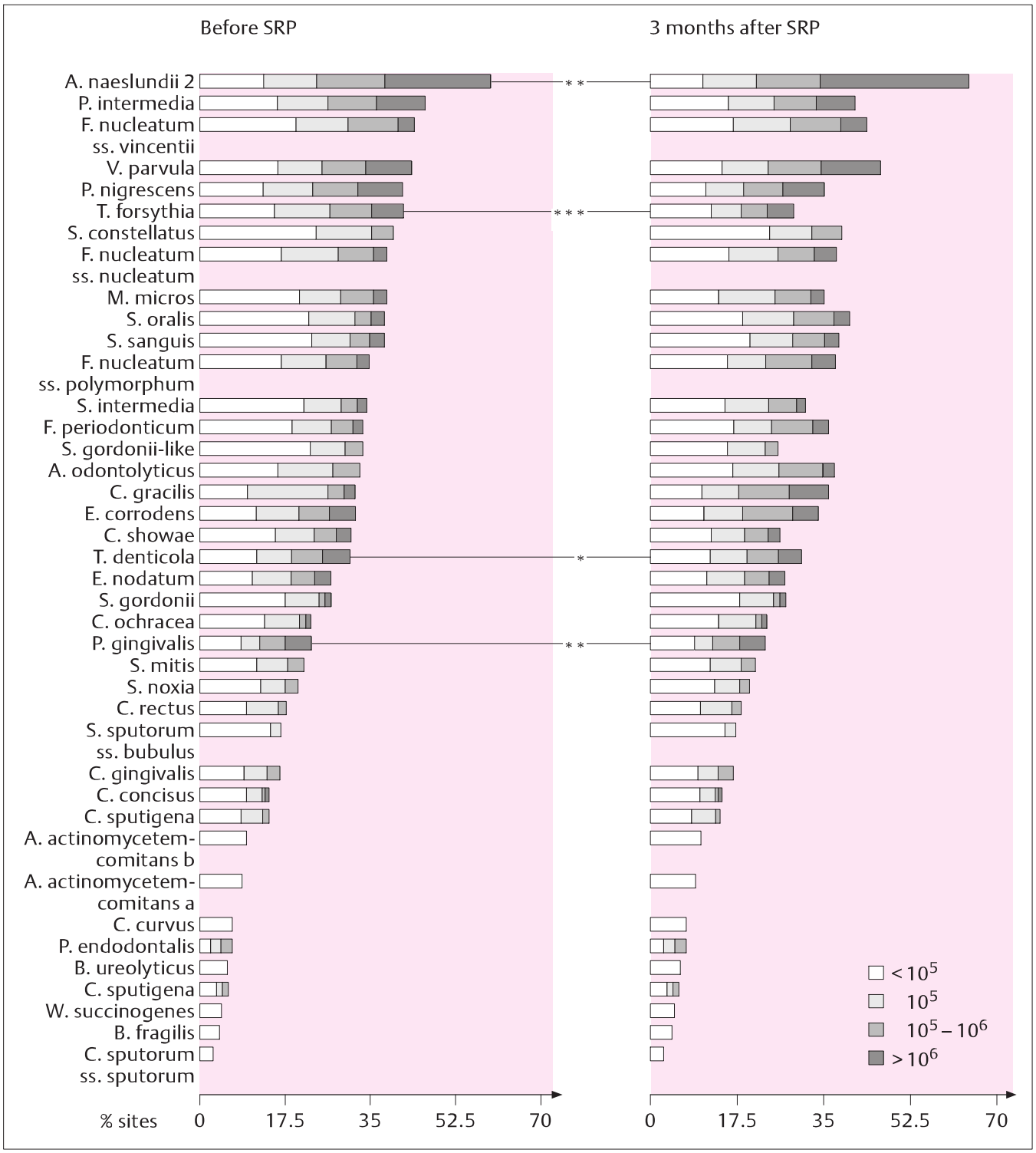


Fig. 10.**8** Alterations in subgingival microflora after subgingival scaling and root planing (SRP) (Haffajee et al. 1997). Prevalences of the 40 most frequent bacterial species of the oral cavity were investigated at each tooth of 57 adult patients with chronic periodontitis. Checkerboard DNA-DNA hybridization was employed. Significant alterations (*: $p < 0.05$, **: $p < 0.01$, ***: $p < 0.001$) occurred for *Actinomyces naeslundii* 2 (↑), *Tannerella forsythia*, *Treponema denticola*, and *Porphyromonas gingivalis* (↓). The prevalence of other bacteria was not significantly changed. Concomitantly, reduction of bleeding after probing and probing depth as well as gain in clinical attachment in pockets of 6 mm or more was registered

One-Stage, Full-Mouth Disinfection

- Compared to the traditional approach of quadrant-wise débridement over several weeks, *oral disinfection* over a short period of time has proved superior by some investigators:
  - When carried out to inhibit recolonization of already débrided root surfaces.
  - Complete definitive supra- and subgingival scaling preferably within 24 hours.
  - In addition, disinfection of all attainable extracrevicular habitats of oral pathogens:
    - Application of chlorhexidine gel into scaled pockets.
    - Continuous and consistent use of chlorhexidine preparations at home for about six weeks: chlorhexidine mouthwash, tooth brushing with chlorhexidine gel, chlorhexidine spray to disinfect the tonsillar region.
    - Tongue cleaning with brushes (chlorhexidine gel) or special scrapers.
    - *Note:* After cessation of chemical plaque control, all staining is to be removed and mechanical plaque control reinforced.

Reevaluation

- About six weeks after subgingival scaling, the periodontal conditions are reevaluated:
  - Periodontal probing depths and clinical attachment levels are assessed.
  - Decisions are made regarding further therapeutic measures.
  - An oral hygiene check is carried out.

Sharpening of Instruments

General Remarks

- Apart from the operator's skill, one important prerequisite for success is sharp instruments:
  - Dull instruments polish subgingival calculus rather than remove it. Subsequent removal is almost impossible.
  - There is an increased risk of fracture of a dull instrument because higher pressure is exerted on it.
  - For this reason, instruments must be regularly sharpened with, e.g., Arkansas or ceramic sharpening stones. Oil or water is used as lubricant.
- Instrument sharpness must be assessed after cleaning and sterilization.
  - Light is reflected on the cutting edge of a dull instrument.
  - Plastic sticks can be used to test sharpness.
  - *Note:* Maintaining the original design of the instrument is mandatory.
- After sharpening, instruments are sterilized again, and the cassettes wrapped.
  - Usually, specially trained nurses should sharpen all the instruments.
  - *Note:* Sharpening of dull instruments shortly before the treatment session or even in front of the patient must be avoided.
- The terminal shank of the instrument, i.e., the area between the blade and the first angle, is a key feature for each scaler or curette (Fig. 10.**9**). Proper alignment of the terminal shank will automatically place the instrument in the correct position for sharpening.

Sickle Scalers

- The instrument with its tip toward the body is held with the nondominant hand and a secure palm grasp. The top shank is braced with

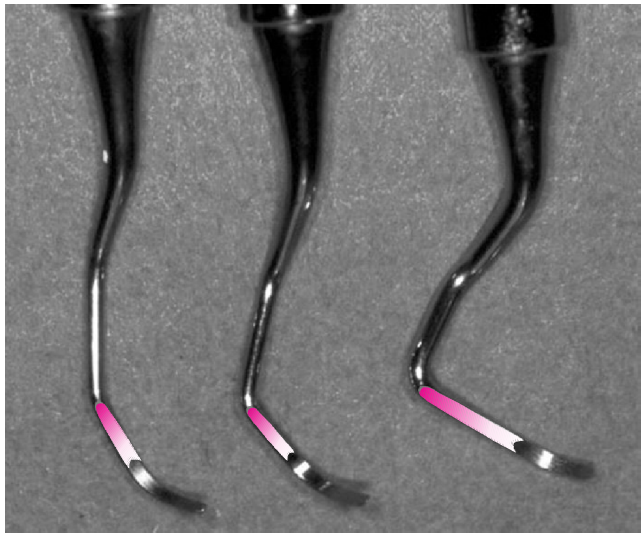


Fig. 10.**9** The terminal shank (*red*) is a key feature of any scaler and curette. Proper alignment of the terminal shank will automatically place the instrument in the correct position for sharpening

the index finger to counterbalance the pressure. The elbow is rested on the table.
- The terminal shank is held in an upright position (0°, 12 o'clock).
- The lower half of the lubricated sharpening stone (oil for Arkansas stone, water for ceramic stone) is grasped by the dominant hand. It is pressed against the right blade of the scaler at an angle of slightly less than 30° (one o'clock for right-handers, 11 o'clock for left-handers).
- The stone is moved up and down with moderate pressure:
  - first against the lateral surface of the heel of the blade;
  - then gradually moving forward to grind the middle part;
  - finally, the stone is advanced to the tip third of the blade.
- The tip of the scaler is then rotated away from the body with the terminal shank upright. The sharpening stone is pressed at an angle of slightly less than 30° to the blade (one o'clock for right-handers, 11 o'clock for left-handers). The heel, middle, and tip thirds are gradually sharpened by moving the stone up and down with moderate pressure.
- Finally, any wire edges may be removed from the face of the scaler with a conical sharpening stone.
- Sharpness is tested with a plastic stick.
  - The stick is held upright with the thumb and the index finger of the nondominant hand.
  - The scaler is held with the dominant hand in a modified pen grasp.
  - The blade of the scaler is slightly tilted at 11 o'clock. The blade must bite into the plastic stick. A metallic sound may be heard.

Universal Curettes

- The same principles apply for universal curettes as for scalers. Note that the rounded toe of the curette has also to be sharpened.
- The terminal shank is held upright (0°, 12 o'clock).
- The lubricated sharpening stone is pressed against the right blade of the curette at an angle of slightly less than 30° (one o'clock for right-handers, 11 o'clock for left-handers).
- The stone is moved up and down with moderate pressure, starting at the heel third of the blade. The stone is continuously moved along the entire length of the blade.
- The curette is rotated so that the toe now points away from the body. The terminal shank is held upright.
- The sharpening stone is tilted at an angle of slightly less than 30° to the blade (one o'clock for right-handers, 11 o'clock for left-handers).
  - Grinding motion is activated at the heel third of the blade.
  - Up and down movements proceed to the middle, then the toe third of the curette.
- To maintain the rounded shape of the toe it is held horizontally (90°, three o'clock for right-handers, nine o'clock for left-handers). The position of the stone is about 30° to the toe (two respectively 10 o'clock). The stone is moved in a slight and consistent up and down motion, rotating around the toe.
- Any wire edges are removed from the face of the curette with a conical stone.
- Sharpness is tested with a plastic stick. The stick is held upright, whereas the blade of the curette is slightly tilted at 11 o'clock. The blade has to bite into the plastic stick. A metallic sound may be heard.

Area-Specific Curettes

- Area-specific curettes have only one cutting edge. The face of the blade is honed at the terminal shank at 70°.
  - The instrument is held with a firm palm grasp, the right lateral surface being the cutting edge.
  - For all odd numbered Gracey curettes the toe points to the body, for even numbered curettes it points away from the body.
  - The terminal shank is tilted to the left by 30° (slightly less than to 11 o'clock).
  - The lubricated stone is positioned to the right lateral surface (blade) and tilted to the right by 30° (one o'clock).
- The stone is moved up and down with moderate pressure, starting at the heel third of the blade. The stone is continuously moved along the entire length of the blade.
- To maintain the rounded shape of the toe it is held horizontally (90°, three o'clock for

right-handers, nine o'clock for left-handers). The position of the stone is about 30° to the toe (either two or 10 o'clock). The stone is moved in a slight, regular up and down motion, rotating around the toe.

- Finally, any wire edges are removed from the face of the curette with a conical stone.
- To sharpen the blade of the corresponding working end, the curette is turned. The toe now points in the opposite direction.
- To test sharpness the plastic stick is held upright. The terminal shank of the blade is held parallel to the stick. The cutting edge is pressed against the test stick. It must bite into the stick. A metallic sound may be heard.

11 Phase II: Corrective Procedures

Periodontal Surgery

General Remarks

- Surgical procedures during periodontal treatment have the following objectives:
  - Treatment of persisting periodontal lesions under visual control.
  - In some cases, alteration of tooth morphology and/or morphology of the gingiva and the alveolar bone in order to achieve a more physiological form.
  - Attempted regeneration of lost periodontal structures.
- More or less resective procedures are differentiated from regenerative procedures:
  - Gingivectomy/gingivoplasty (resective)
  - Flap operations (resective, sometimes regenerative)
  - Guided tissue regeneration (regenerative).
- Further surgical procedures may be performed during the corrective stage:
  - Plastic periodontal surgery
  - Oral surgical measures
  - Placement of implants
- The definitive restorative treatment should be carried out no earlier than four to six months after surgical measures.
- In some cases, orthodontic treatment is also necessary.

Gingivectomy

General Remarks

- During (external) gingivectomy, all of the pathological tissue is surgically removed while maintaining the physiological form of the gingiva.
- Advantages:
  - Quick and simple technique.
  - Any pockets are eliminated.
- Disadvantages:
  - Radical procedure with a high risk of undesired root exposure; especially in the ante-

Table 11.1 Standard instruments for gingivectomy

Instruments	Description	Article no., manufacturer
Mouth mirror	Plane, front surface rhodium-coated, ∅ 22 mm	M4C, Hu-Friedy
Periodontal probe	Calibration in 1-mm steps or color coded 3–3–2–3 mm	PCPUNC15 or PCP11, Hu-Friedy
Pliers	• Dressing pliers • Periodontal marking pliers, left and right • Anatomical tissue pliers • Surgical tissue pliers	• DP18 or DP17, Hu-Friedy • PMGF1+2, Hu-Friedy • TP31 or TPG1, Hu-Friedy • TP33 or TPG3, Hu-Friedy
Surgical retractors	• Langenbeck retractor • Middeldorpf retractor	• SR2, Hu-Friedy • RSMID2, Hu-Friedy
Scalpel holder	• Straight scalpel holder • Universal 360° blade handle	• 10-130-05E, Hu-Friedy • K360, Hu-Friedy
Scalers	Sickle scaler CI2/3	SCI2/36, Hu-Friedy
Curettes	Universal curettes • Posterior teeth in the lower jaw: Langer 1/2 • Posterior teeth in the upper jaw: Langer 3/4 • Anterior teeth: Langer 5/6	 • SL1/26 or SL12AF, Hu-Friedy • SL3/46 or SL3/4AF, Hu-Friedy • SL5/66 or SL5/6MF, Hu-Friedy
Scissors	• Goldman–Fox gingival scissors • LaGrange gingival scissors	• S16, Hu-Friedy • S14, Hu-Friedy

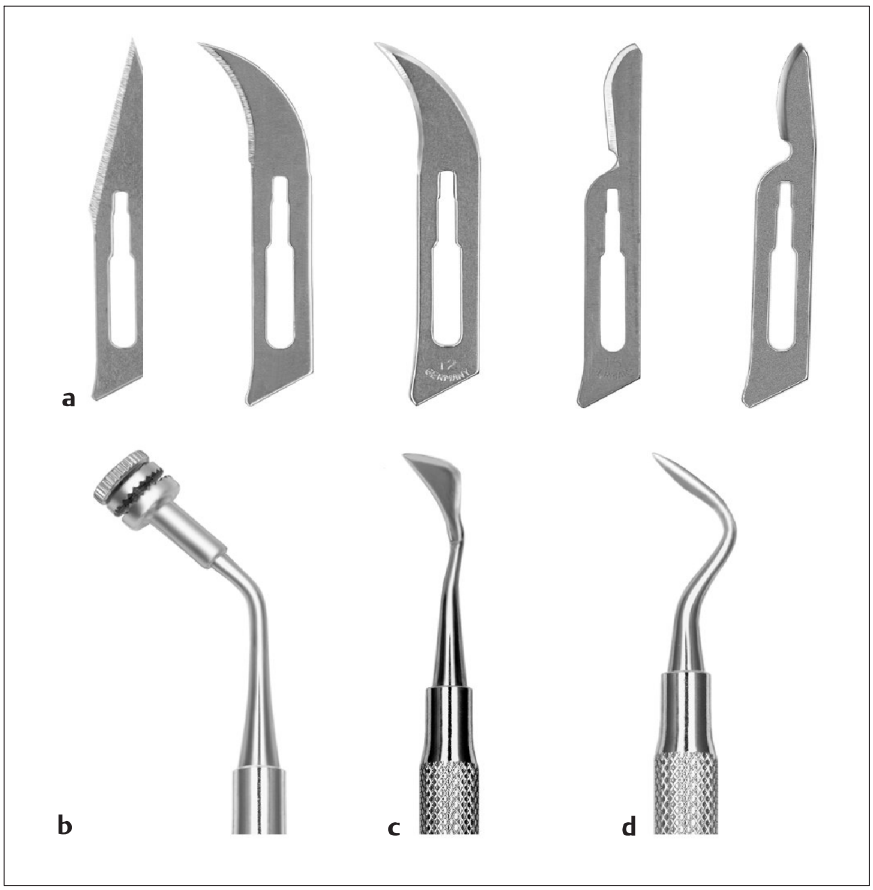


Fig. 11.**1** Scalpel blades and blade handles.
a Disposable scalpel blades (from *left* to *right*): no. 11, no. 12, no. 12D, no. 15, no. 15C.
b Universal 360° blade handle.
c Hatched-shaped Kirland knife.
d Lancet-shaped Orban knife

rior region, there is a risk of esthetic problems arising.
– Increased dentin hypersensitivity.

Aims

- Excision of fibrotically thickened gingiva
- Pocket elimination

Indications

- Indications are quite restricted:
  – Supraalveolar pockets of more than 4 mm in the presence of fibrotic, thickened gingiva; e. g., hereditary gingival fibromatosis or drug-induced gingival enlargements.
  – Preprosthodontically, to expose a subgingival preparation line before making the impression.
  – To surgically expose impacted teeth in cases of eruption anomaly.

Contraindications

- Contraindications exist mainly where there are indications for alternative treatment procedures:
  – Particularly in esthetically sensitive areas, e. g., anterior teeth in the maxilla with a periodontal phenotype characterized by thin and narrow gingiva
  – Infrabony pockets
  – Bulged thickening of the bone margin with the risk of surgical exposure

Instruments (Table 11.**1**)

- Pliers:
  – Special pliers for pocket marking
  – Anatomical and surgical tissue pliers
- Scalpel blades (Fig. 11.**1 a**):
  – No. 11: lancet-shaped
  – No. 12: sickle-shaped, blade on one side only; no. 12D: blades on both sides
  – No. 15, no. 15C: rounded

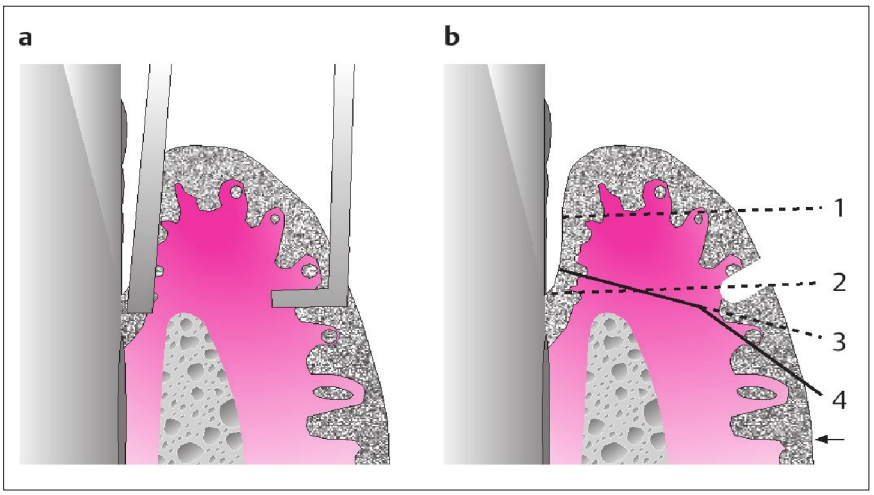


Fig. 11.**2** External gingivectomy.
a Bleeding points are set with pocket marking pliers.
b Incision lines *3* and *4* are recommended for gingivectomy. Incision line *1* does not remove the pocket, while line *2* leads to balcony-like contour. Mucogingival border (⟵) and alveolar bone should not be involved

- Scalpel handle (Fig. 11.**1 b**):
  - Straight blade handle
  - Universal 360° blade handle
- Gingivectomy knives (only for local gingivectomy), e.g.:
  - Hatchet-shaped Kirkland knife (Fig. 11.**1 c**)
  - Lancet-shaped Orban knife (Fig. 11.**1 d**)
- Scissors:
  - Goldman–Fox gingival scissors
  - LaGrange gingival scissors

Procedure

- External gingivectomy consists of the following *treatment steps:*
  - Disinfection, e.g., mouth rinsing with povidone-iodine or chlorhexidine solution for two minutes
  - Local anesthesia
  - Creating bleeding points with marking pliers
  - Incision
  - Tissue excision
  - Scaling and root planing
  - Recontouring of the gingival margin
  - Cleaning of the wound area
  - Periodontal dressing
- *Bleeding points* are produced with special marking pliers (Goldman–Fox or Crane–Kaplan):
  - One arm of the pliers is inserted into the pocket, while the one with the sharp inward point remains on the outside (Fig. 11.**2 a**).
  - By pinching the pliers, a bleeding point is set on the gingiva, marking the level of the bottom of the pocket.
- *Incision:*
  - Nowadays disposable scalpel blades are usually used. *Note:* In lingual and distopalatal areas, incisions cannot be made with straight scalpel handles.
  - No. 11 or 12D scalpel blades are fixed in the universal 360° handle perpendicular to the handle.
  - Gingivectomy knives are only used for small, regionally limited interventions. Disadvantage: blades must be sharpened after every use.
  - Continuous incision across the entire surgical site (Fig. 11.**3**):
    - Incision angle to the tooth axis of about 120° (Fig. 11.**2 b**, incision line *3*).
    - Incision ends apical to the bleeding points.
    - A horizontal incision is not recommended (Fig. 11.**2 b**, incision lines *1* and *2*) because of the resulting balcony-like contours.
    - Exposure of the bone should be avoided because it leads to delay in wound healing, attachment loss, and pain.

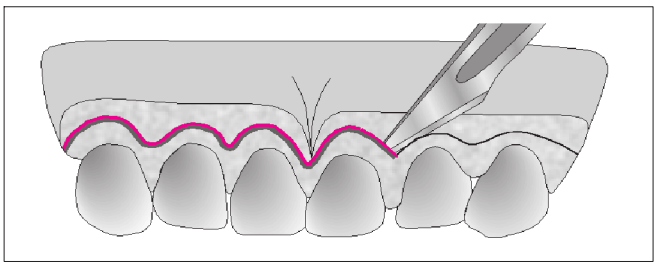


Fig. 11.**3** Gingivectomy. Continuous scalloped incision at an angle of 120° to the tooth axis

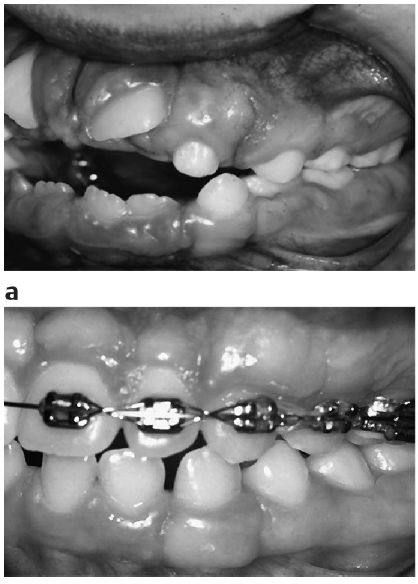
- Interdental separation and excision of the tissue may be carried out with the CI2/3 scaler. Recontouring of the incision edge may be done with gingival scissors (Fig. 11.**2 b**, incision line *4*).
- Careful scaling and root planing are performed.
- Any tissue remains is removed with gauze soaked in physiological saline.
- In all cases a soft periodontal dressing (CoePak) must be applied, which should be renewed after one week.
- In cases of full-mouth gingivectomy (which should be performed with the patient under general anesthesia), a vacuum-formed acrylic splint may serve as a tray for the periodontal pack.

Postoperative Care

- Mouth rinsing to control infection postoperatively is performed twice daily with a 0.1–0.2 % chlorhexidine solution until normal oral hygiene measures become possible.
- Periodontal dressing is removed after one week:
  - The wound surface is cleaned and fibrin and desquamated epithelial cells removed.
  - Usually, a new periodontal dressing is applied.

Critical Assessment

- Postoperative complications are more frequent after gingivectomy procedures than after other periodontal surgical interventions:
  - Secondary wound healing generally requires a periodontal dressing (CoePak).
  - Epithelialization starts from the incision edges. Thus, interdental areas are epithelialized last, i. e., after 10–14 days.
- Care should especially be taken in esthetically sensitive areas. Be aware of possible contraindications regarding anterior teeth in the maxilla.
- Nevertheless, the impression that this is an outdated technique may be wrong:
  - Periodontal phenotypes have to be considered (cf. Fig. 7.**7**). Extremes are:
    - Highly-scalloped, thin, and narrow gingiva which tends to recede after traumatic insult or chronic inflammation. In such cases a gingivectomy is absolutely contraindicated.
    - Wide and thick gingiva, usually associated with rather square anterior teeth in the maxilla. This phenotype tends to pocket formation and represents a possible indication for gingivectomy.
    - *Note:* The periodontal phenotype is genetically determined and cannot be altered by surgical procedures.



a

b

c

Fig. 11.**4** **a** Eight-year-old child with hereditary gingival fibromatosis and severe eruption anomaly of permanent teeth.
b Following gingivectomy and extraction of the deciduous lateral incisors in the maxilla, orthodontic treatment commenced.
c Three years after commencement of orthodontic therapy. Although gingivectomy procedures have been repeated, gingiva remains thick and bulgy

– Gingivectomy is quite frequently indicated in patients with drug-induced gingival enlargement (calcium antagonists, cyclosporine, phenytoin).

➤ Successful gingivectomy depends on strict observance of indications and contraindications. *Note:* In cases of hereditary gingival fibromatosis, recurrent enlargement is to be expected, especially during body growth (Fig. 11.**4**).

➤ Gingivectomy may also be conducted with pulsed infrared lasers (Nd:YAG or CO_2 laser).
– It is questionable whether this provides any advantage over gingivectomy performed with a scalpel.
– It could be an important option for hemophilic patients and patients receiving anticoagulation therapy.
– Reflection of laser light from metallic restorations and metal instruments must be avoided.

Gingivoplasty

Definition

➤ Minor plastic surgical correction of the gingiva in order to alter its contour.

Aim

➤ To reshape the gingiva in order to obtain a more physiological contour.

Indications

➤ Gingivoplasty, which is a regionally limited intervention, may be indicated in case of:
– Regionally limited thickening of the gingiva without the presence of pathologically deepened pockets.
– Persistent interdental craters after necrotizing ulcerative gingivitis/periodontitis.
– As an additional step during gingivectomy (cf. Fig. 11.**2 b**, incision line *4*): smoothing of the incision edge to avoid balcony-like contours.
– Preprosthodontically:
• To reshape the gingiva in the area of the later pontic.
• To expose preparation lines before impression making.

Contraindications

➤ Consequently, contraindications are:
– Generalized bulgy and thickened, fibrous gingiva.
– Periodontal pockets.

Instruments

➤ Gingiva may be carefully modeled with:
– Scalpel (blade no. 11 or no. 12D)
– LaGrange or Goldman–Fox gingival scissors
– Diamond-coated burs
– Electrosurgically.

➤ Dental electrosurgical devices have a power of 50 W:
– Monoterminal use; no neutral electrode is required.
– Current setting "rectified and filtered" (nonmodulated high-frequency current, about 2 MHz).
– *Note:* Strongly modulated high-frequency current for electrocoagulation, electrofulguration, or electrodesiccation is not used in dentistry. *Warning:* Avoid injuries of
• the pulp, e. g., by touching metal restorations;
• cementum;
• periodontal ligament;
• bone.

➤ For preference, needle-shaped, rhomboid, elliptic, or round sling electrodes are used (Fig. 11.**5**).

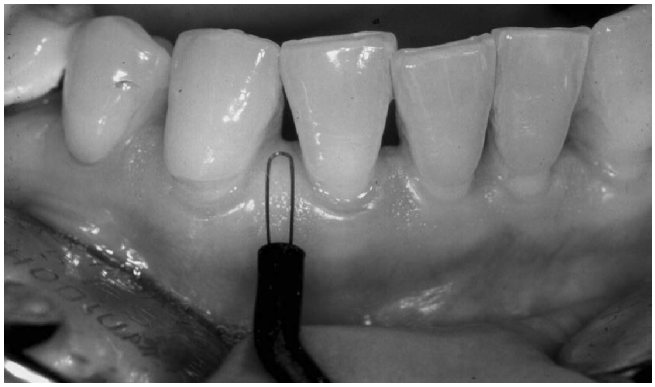


Fig. 11.**5** Sling electrode for electrosurgical contouring of gingiva

Procedure

- Fast, decisive working is recommended. Only the superficial surface of the tissue is to be removed.
  - *Note:* Heat development depends mostly on the length of time the electrode is moved through the tissue.
  - Electrical spark formation represents extreme energy density and leads to carbonization.
  - Contact with tooth or, especially, bone *must* be avoided.
  - Smoke formation has disastrous psychological effects, so make sure of excellent suction.

Critical Assessment

- *Advantages* of electrosurgery are:
  - tiny electrodes, no sharpening required;
  - ischemic conditions because of immediate coagulation of small vessels and capillaries;
  - hence, excellent visual control.
- *Disadvantages*, however, which in the end outweigh the advantages, are:
  - high risk of deep tissue injury;
  - risk of postoperative infection, delayed wound healing, sequestrum formation;
  - unpleasant smell.
- *Note:* Electrosurgical procedures do not play an important role in periodontal surgery.
- Preprosthodontically, gingiva might be modeled electrosurgically in the area of the later pontic and for exposure of preparation lines:
  - *Note:* Before making an impression of subgingival preparation lines, the (inflammation-free) gingival margin should be carefully retracted with a retraction cord rather than exposing the lines electrosurgically.
  - Unlike electrosurgery, this procedure does not lead to attachment loss.

Flap Operations

Aims

- Periodontal flap surgery is performed for the following reasons:
  - To create access to the infected root surface when the morphology of bony lesions is complicated and there is furcation involvement.
  - To facilitate careful débridement of root surfaces under visual inspection.
  - To surgically alter unfavorable morphology of the alveolar bone (osteoplasty) or tooth (odontoplasty).
  - To regenerate lost periodontal tissues.

Indications

- Flap operations may be indicated in the following cases:
  - Pockets deeper than 5 mm persisting after phase I therapy
  - Especially, bony pockets and interdental craters
  - Bony lesions in the region of furcations
  - Need for surgical crown lengthening
- *Note:* When to open up a flap on a periodontal lesion with complicated morphology is mainly a question of technique, e.g.:
  - When it is difficult to insert a curette to the bottom of the bony lesion in a deep and narrow pocket
  - When it is difficult to achieve the correct angle of the curette blade against the root surface
  - Loss of control while working on the root surface in deep pockets

Contraindications

- Flap operations are not indicated in the following cases:
  - For shallow, supraalveolar pockets, especially in esthetically sensitive areas, when subgingival scaling and root planing can easily be repeated
  - On fibrous, thickened gingiva, where gingivectomy might result in more favorable tissue morphology

Instruments

- In addition to the surgical instruments already mentioned, the following instruments are used (Table 11.**2**):
  - An appropriately small elevator for mobilization of the mucoperiosteal flap

Table 11.2 Standard instruments of tray for periodontal flap surgery

Instruments	Description	Article no., manufacturer
Mouth mirror	Plane, front surface rhodium-coated, ∅ 22 mm	M4C, Hu-Friedy
Periodontal probe	Calibration in 1-mm steps or color coded 3–3–2–3 mm	PCPUNC15 or PCP11, Hu-Friedy
Pliers	• Dressing pliers • Anatomical tissue pliers • Surgical tissue pliers	• DP18 or DP17, Hu-Friedy • TP31 or TPG1, Hu-Friedy • TP33 or TPG3, Hu-Friedy
Surgical retractors	• Langenbeck retractor • Middeldorpf retractor	• SR2, Hu-Friedy • RSMID2, Hu-Friedy
Scalpel holder	• Straight scalpel holder • Universal 360° blade handle	• 10-130-05E, Hu-Friedy • K360, Hu-Friedy
Periosteal elevator		P24GSP or P8D, Hu-Friedy
Scalers	Sickle scalers CI2/3 and T2/3	SCI2/36 or ST2/36, Hu-Friedy
Curettes	Universal curettes Goldman–Fox 4 Area-specific curettes • Anterior teeth: Gracey 1/2 or 5/6 • Buccal/lingual surfaces of posterior teeth: Gracey 7/8 • Mesial surfaces of posterior teeth: Gracey 11/12 or 15/16 • Distal surfaces of posterior teeth: Gracey 13/14 or 17/18	SGF4, Hu-Friedy • SG1/26 or SAS1/2, Hu-Friedy • SG7/86 or SAS7/8, Hu-Friedy • SG11/126 or SRPG11/126, or SRPG15/16, Hu-Friedy • SG13/146 or SRPG13/146, or SRPG17/186, Hu-Friedy
Bone files	• Schluger • Sugarman	• FS9/10S, Hu-Friedy • FS1/2S, Hu-Friedy
Scissors	• Goldman–Fox gingival scissors • LaGrange gingival scissors	• S16, Hu-Friedy • S14, Hu-Friedy
Needle holder	Olson–Hegar	NH5068, Hu-Friedy
Suture material	• C6 reverse cut, 3/8 circle, 4-0 polyester or polypropylene • C3 reverse cut, 3/8 circle, 5-0 polyester or polypropylene	• PSNR683L or PSN8683P, Hu-Friedy • PSNR698L or PSN8698P, Hu-Friedy

  - Universal curettes for removal of granulation tissue, particularly from bony pockets and furcations
  - Needle holders and suture material
- ➤ Some supplementary instruments, which are not regularly used, should be kept at hand:
  - Sugarman and/or Schluger bone files for osteoplasty.
  - Special furcation curettes.

Various Techniques

- ➤ Surgical techniques employing flap operation were first described between 1912 and about 1920 by R. Neumann, A. Cieszynski, and L. Widman.
  - They were generally described as a radical operation regularly involving bone resection.
  - As a rule, the bottom of the bony pocket became the new alveolar crest.
- ➤ In 1931 Kirkland described a modified flap operation: only interdental papillae were elevated, and they were carefully replaced and sutured after root débridement.
- ➤ During the 1950s and 1960s, radical methods were propagated again, e.g., the *apically repositioned flap* (Nabers 1954, Friedman 1955, 1962):

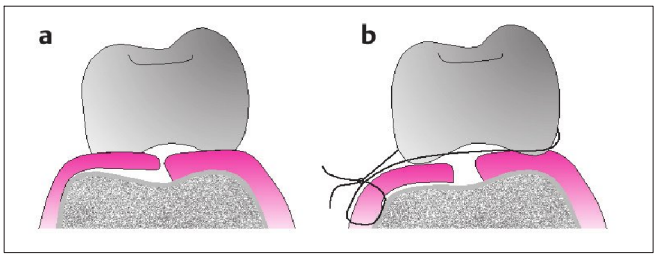


Fig. 11.**6** Periosteal suture for fixation of the buccal flap in an apical position. Note that some bone has been surgically removed buccally (surgical crown lengthening)

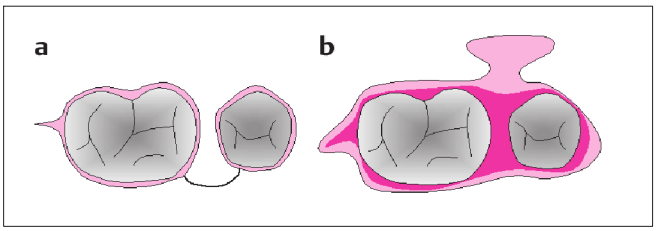


Fig. 11.**7** Incision lines for a papilla preservation flap (Takei et al. 1985). Semilunar circumcision of the papilla palatally. Mobilization of the papilla in a buccal direction. Inflammation-free gingiva and adequate width of the papilla (>2 mm) are prerequisites

- After bone resection, gingiva was apically repositioned and fixed by periosteal sutures.
  - Advantage: pockets were totally eliminated; however, in contrast to gingivectomy, keratinized tissue was preserved.
  - Disadvantage: considerable bone loss after leveling of bony pockets.
- Nowadays, this technique is performed only where careful osteoplasty is required, e. g.:
  - Surgical exposure of impacted teeth
  - Tunnel preparation of mandibular molars where the furcation is involved
  - Surgical crown lengthening
- In most cases only buccal flaps are apically repositioned; on the lingual/palatal aspects either gingivectomy is performed or the flap is shortened:
  - Elevation of the mucoperiosteal flap
  - Careful osteoplasty with, e. g., universal curettes (Columbia 4R/4L) and/or bone files (Schluger, Sugarman)
  - Meticulous root planing to prevent undesired reattachment
  - Fixation of the flap with periosteal sutures in its new position (Fig. 11.**6**).

➤ A *papilla preservation flap* was described by Takei et al. (1985) to cover interdentally placed graft material, e. g., for:
- Implantation of bone substitutes.
- Guided tissue regeneration.
- Prerequisites: wide (>2 mm) and inflammation-free interdental gingiva
- The papilla is circumcised palatally/lingually and mobilized in a buccal direction (Fig. 11.**7**).
- The buccal periosteum must be dissected to advance the flap coronally and cover the graft material. The palatal papillae are at high risk of becoming necrotic. For this reason, the method was modified by Cortellini et al. (1995) (cf. Fig. 11.**21**):
  - Semilunar circumcision of the papilla buccally.
  - Coronal mobilization of the vestibular flap after dissection of the periosteum at its base.
  - This may allow coverage of, e. g., a space-keeping membrane.

➤ A widely used technique is the *modified Widman flap* (Ramfjord and Nissle 1974):
- Standard technique to create access to bony lesions (access flap).
- Soft tissue is largely preserved.
- Advantages:
  - Excellent view of the surgical site.
  - Instrumentation under direct visual control is possible.
  - Correct suturing may allow primary wound healing.

Procedure for the Modified Widman Technique

➤ This flap operation consists of the following steps:
- Disinfection
- Local anesthesia
- Placing of several incisions (Fig. 11.**8**), preferably with a 12D scalpel blade:
  - *Paramarginal incision* up to the bone crest, particularly palatally in cases of fibrous, thickened gingiva (reverse bevel incision; sometimes called *internal gingi-*

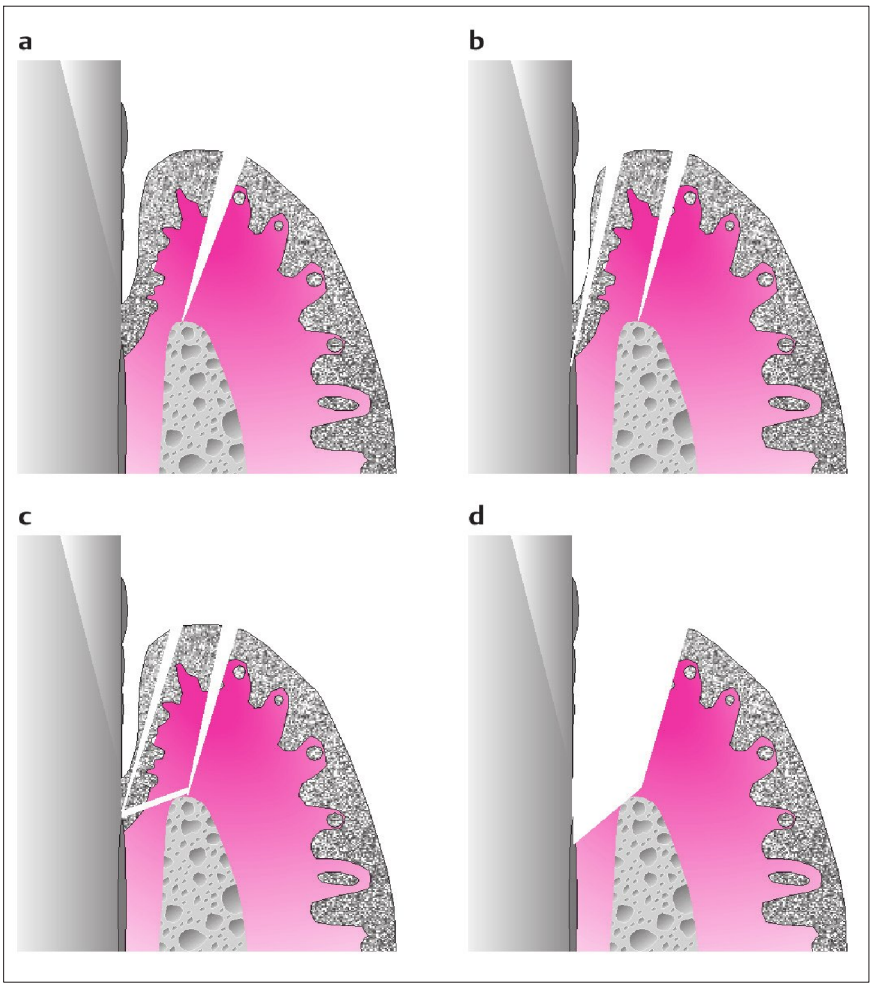


Fig. 11.**8** Incisions of the modified Widman flap technique.
a Paramarginal incision (reverse bevel incision).
b Intracrevicular incision to the bottom of the defect.
c Incision that is as near horizontal as possible. The granulation tissue has now been circumcised and can be removed (**d**)

vectomy) (Fig. 11.**8 a**). *Note:* Paramarginal incisions should be avoided in esthetically sensitive areas and in patients with thin gingiva.
  - *Intracrevicular incision* up to the bone crest (Fig. 11.**8 b**).
  - *Horizontal incision* (perpendicular to the tooth axis) after careful mobilization of the flap (Fig. 11.**8 c**).
  - In cases of isolated bony pockets, one or two vertical releasing incisions may be necessary, which bound the surgical site laterally: the incisions should be slightly paramedian and diverging into the vestibulum to avoid postoperative recession or papilla necrosis.
- Careful mobilization of a full mucoperiosteal flap with an elevator.
- Removal of circumcised tissue with the CI2/3 sickle scaler.
- Curettage of granulation tissue with GF4 universal curette (Fig. 11.**8 d**).
- Scaling and root planing with area-specific curettes and, if appropriate, periodontal files (Orban, Hirschfeld).
- Removal of granulation tissue from the inner surface of the flap with gingival scissors, e. g., LaGrange.
- Fixation and suturing of the flap in its original position:
  - This may only be possible if the flap is not mobilized beyond the mucogingival border; otherwise it will collapse at the alveolar crest.
  - In order to sufficiently close the wound, coronal displacement of the buccal flap after dissection of the periosteum at its base may be necessary.
- Suturing:

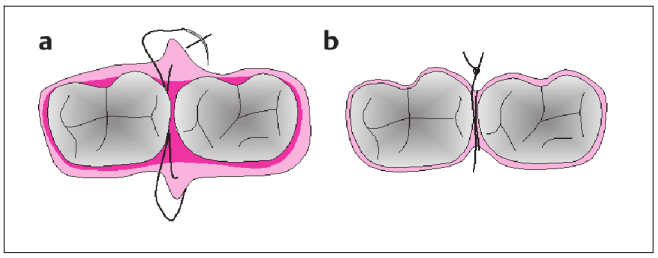


Fig. 11.**9** **a** Simple interrupted suture for fixation of the flap in its original position.
b If no periodontal dressing is planned, knots are generally placed buccally (lingual placement is very disturbing for the patient's tongue). If a periodontal dressing is to be placed, knots are placed lingually/palatally. For suture removal, the buccal part of the dressing is removed first. Sutures are cut with scissors. The palatal part of the dressing embedding the knots can then easily be removed together with the sutures

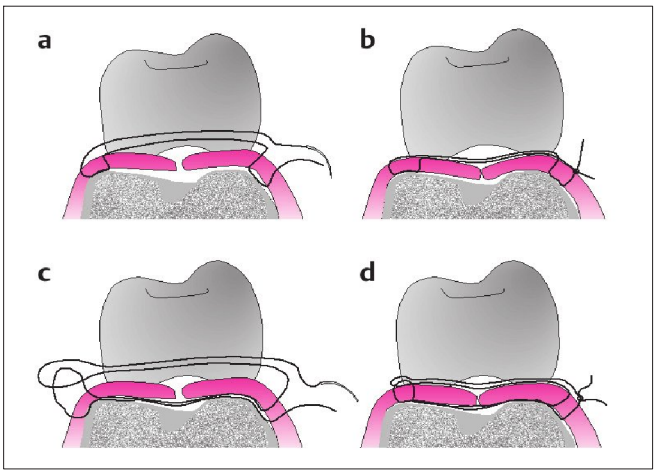


Fig. 11.**11** Vertical mattress suture.
a, b Simple vertical mattress suture.
c, d Vertical mattress suture additionally secured

- In general, synthetic suture material should be used: atraumatic material, 4-0 or 5-0. If sutures are placed for more than 10–14 days, monofil polypropylene or polytetrafluoroethylene is recommended.
- Interrupted sutures (Fig. 11.**9**), e. g., with C6 needle, reverse cut, three-eighths of a circle, atraumatic, 4-0. About six sutures may be placed with one thread of 45 cm.
- A double knot (friction knot) is placed first, followed by a single knot in the other direction with another single on top in the same direction as the first (double) knot.
- If a periodontal dressing is necessary, all knots should be placed lingually, which makes removal of dressing and sutures easier; otherwise, all knots should be placed buccally.

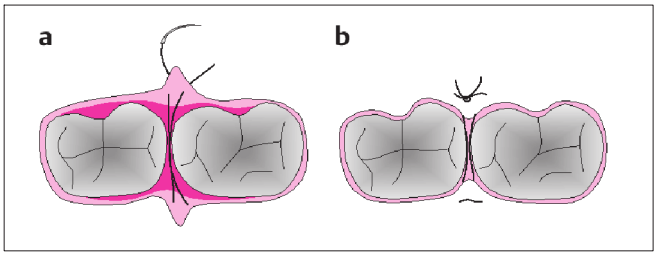


Fig. 11.**10** Horizontal mattress suture for tight interdental wound approximation

- Finally, vertical releasing incisions, if any, are sutured (C3, 5-0).
- Horizontal (Fig. 11.**10**) and vertical *mattress sutures* (Fig. 11.**11**) are used to tightly approximate the interdental wound margins.
- Some operators prefer *continuous sutures* (Fig. 11.**12**). A C6 needle is used. The suture starts from the anterior region and secures first the buccal flap with tooth-embracing sling sutures. Then, sling sutures also secure the lingual flap. Only one anterior knot is placed.

– If needed, a periodontal dressing (CoePak) is placed to protect the surgical site.

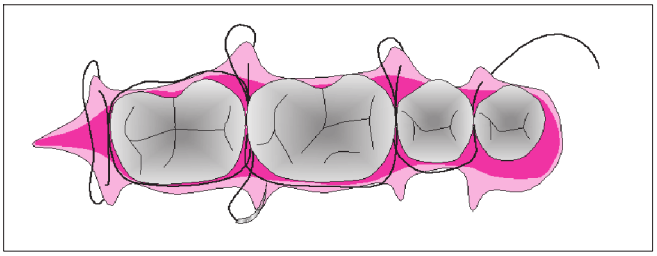


Fig. 11.**12** Continuous suture. The suture starts in the anterior region of the surgical site in the first buccal papilla. Tooth-embracing sling sutures secure the buccal flap. Continuing distal to the most posterior tooth, the palatal/lingual flap is now also secured with sling sutures. There is only one anterior knot which is placed buccally

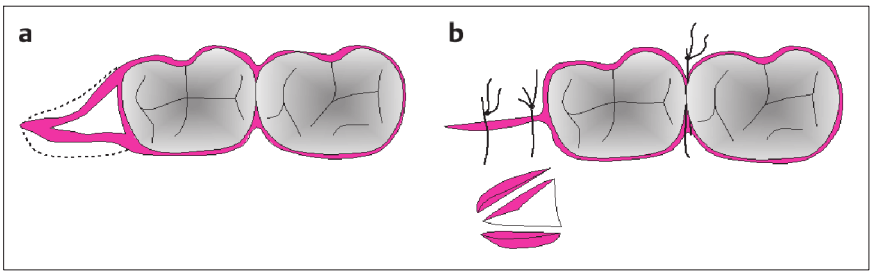


Fig. 11.**13** Distal wedge operation with excision of fibrotic, thickened gingiva, e.g., in the maxillary tuberosity. A central wedge is first circumcised (**a**) and removed. Palatal and, if needed, buccal flaps are thinned with the scalpel, resulting in a further one or two wedges. **b** After removal of these wedges and thorough root planing, the flaps are tightly approximated to the alveolar crest

- *Wedge operation:* frequently performed as a distal wedge in cases of fibrotic and thickened gingiva at the maxillary tuberosity, sometimes with distal furcation involvement of the most posterior molar (Fig. 11.**13**). Also preprosthodontically for crown lengthening of the wisdom tooth or second molar:
  - Circumcision and removal of a tissue wedge distal to the last tooth.
  - Sharp preparation and removal of additional palatal and, if needed, buccal wedges (internal gingivectomy).
  - Careful instrumentation of the root surface, particularly in the distal furcation area.
  - Tight approximation of the flaps to the alveolar crest with interrupted sutures (cf. Fig. 11.**9**) or, if the surgical site is more extended, continuous suture (cf. Fig. 11.**12**).

Postoperative Care

- If interdental wound closure can be achieved, periodontal dressing is usually unnecessary.
- Periodontal dressing should protect the wound area from chemical, thermal, and mechanical insult during healing. Further indications for a dressing may be, for example:
  - Psychological reasons.
  - Stabilization of highly mobile teeth.
- A periodontal dressing should have the following properties:
  - Applied in a soft condition and sets rapidly.
  - Sufficiently firm after setting.
  - Smooth surface.
  - Does not interfere with wound healing.
  - Antimicrobials can be added if necessary.
- The following periodontal dressings may be used:
  - Nobetec: based on zinc oxide and eugenol; very firm periodontal dressing.
  - PeriPac: calcium sulfate, sets if exposed to saliva.
  - CoePak: eugenol-free synthetic dressing, which remains quite soft after setting and has a very smooth and pleasant surface.
  - Light-curing dressings, e.g., Barricaid; can be applied without pressure.
- Postoperative infection control:
  - The usual mechanical oral hygiene measures should be practiced in nonoperated areas.
  - In addition, chemical plaque control is necessary so long as effective oral hygiene with toothbrushes is not possible in the wound area, i.e., for about four to six weeks: mouth rinsing with 0.1–0.2% chlorhexidine solution for one to two minutes, twice daily.
  - The patient should be informed about possible, usually mild, adverse effects:
    - Black staining of the dorsum of tongue (hairy tongue)
    - Discoloration of teeth and restorations
    - Taste alterations
    - Infrequently, epithelial desquamation
- Suture removal after 7–10 days:
  - If a periodontal dressing was placed, a second one is usually unnecessary.
  - Suture removal may be difficult after continuous (cf. Fig. 11.**12**) and secured vertical mattress suture (cf. Fig. 11.**11 c**, **d**).
  - *Note:* After implantation of foreign material (bone substitute, membrane for guided

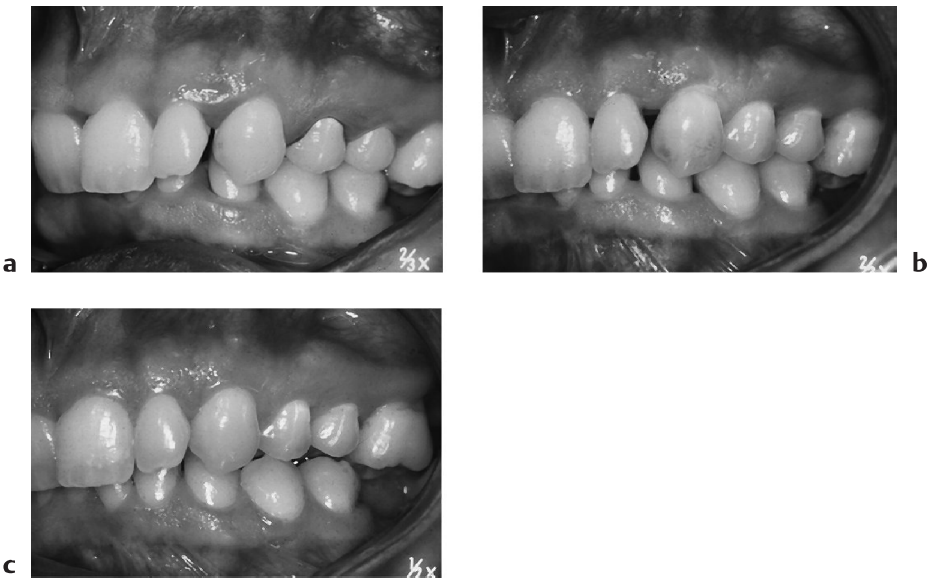


Fig. 11.**14** Particularly in young patients, considerable regeneration of soft tissue can be expected after flap surgery.
a A 28-year-old woman with advanced periodontitis.
b Situation six weeks after surgical therapy. Considerable recession of the gingiva; note particularly loss of the papilla between the lateral incisor and the canine in the maxilla.
c Situation after three years. Gingiva shows no sign of inflammation. Note complete regeneration of papillae

tissue regeneration) or root coverage, sutures should remain in place for an extended period. In these cases use of monofil polypropylene or polytetrafluoroethylene is recommended.

➤ Further follow-up inspections should be conducted every second week until reevaluation after two to three months.

Critical Assessment

➤ Following flap surgery, the inflammatory infiltrate in the gingiva will be greatly reduced.
– The tendency to bleed upon probing is reduced and gingival swelling goes down.
– Some gingival recession may lead to esthetic deficits and dentinal hypersensitivity, but these are not usually too much of a problem (Fig. 11.**14**).

➤ The resistance of the soft tissues to probing pressure increases. The periodontal probe cannot be inserted between tooth and gingiva as deep as before therapy:
– Reduction of periodontal probing depth.
– Apparent, i.e., clinical, attachment gain (Fig. 11.**15**):
• *Note:* Clinical attachment gain is related to the number of bony walls of the periodontal lesion: the more walls, the better the result.

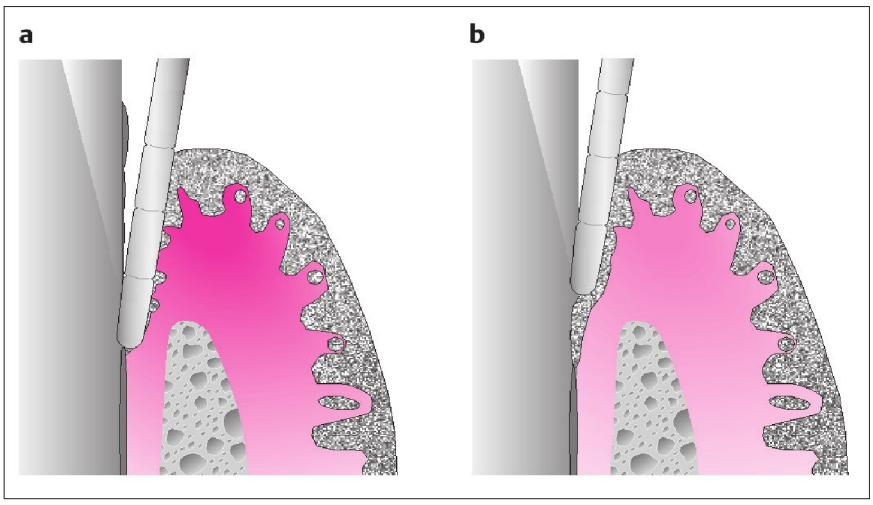


Fig. 11.**15** Clinical attachment gain after flap surgery.
a Situation before surgery. Periodontal probe passes the remains of junctional epithelium at the bottom of the pocket and is stopped only by the supraalveolar connective tissue.
b After treatment, the inflammatory infiltrate has disappeared. A long junctional epithelium has formed, which attaches to the root surfaces at about the same level as the bottom of the former periodontal lesion. At an appropriate probing force of about 0.25 N, the probe cannot pass the entire long junctional epithelium, suggesting some gain of attachment

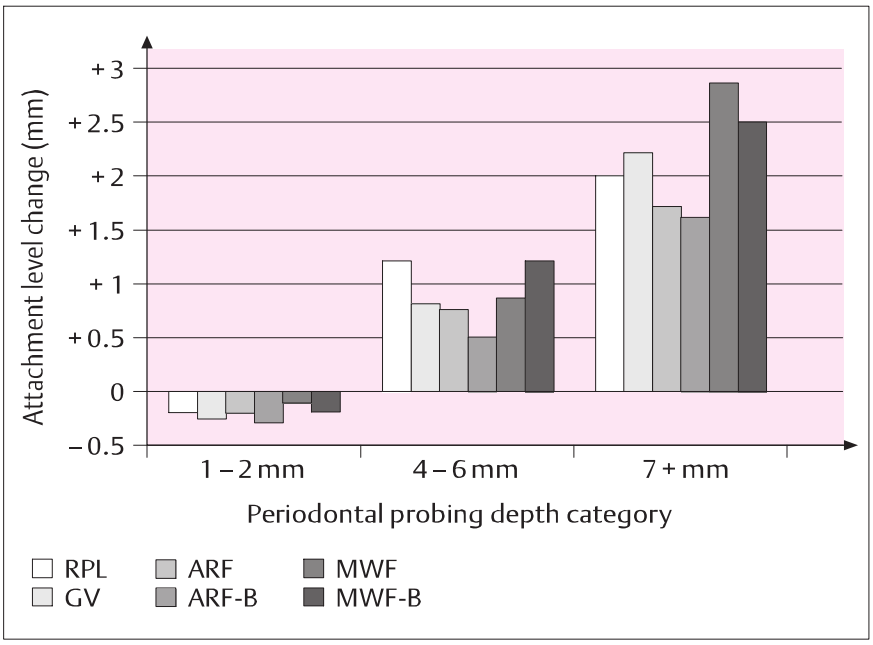


Fig. 11.**16** Average alterations of attachment levels six months after treatment. Results after various treatment modalities: *RPL* scaling and root planing; *GV* gingivectomy; *ARF(-B)* apically repositioned flap (with bone recontouring); *MWF(-B)* modified Widman flap (with bone recontouring). In shallow pockets (up to 3 mm) slight attachment losses were observed. In moderately deep pockets (4–6 mm), attachment gain averaged 0.5–1 mm. In deep pockets (7 mm or more), the modified Widman flap performed best with an average attachment gain of more than 2.5 mm (Westfelt et al. 1985)

- Considerably more clinical attachment gain may be expected in plaque-free dentitions.

➤ Because of mechanical trauma during surgery, teeth become generally more mobile:
- – However, tooth mobility decreases gradually during the healing period.
- – After therapy, teeth should become at least as firm as before surgery.

➤ Postoperative results after flap surgery may be differentiated as follows:
- – Slight attachment loss in shallow pockets.
- – Clinical attachment gain of about 1 mm in 4- to 6-mm-deep pockets
- – Considerable greater clinical attachment gain of more than 2.5 mm, on average, in deep pockets of 7 mm or more (Fig. 11.**16**).
- – No new *connective tissue attachment* is to be expected after conventional flap surgery:
  - Rather, a long epithelial attachment forms.
  - *Note:* In contrast to earlier beliefs, a long junctional epithelium seems *not* to be an area of minor resistance.
- – In intrabony pockets, bone fill can usually be observed:
  - Bone fill cannot be seen radiographically until at least 6–12 months after surgery (Fig. 11.**17**).
  - The more bony walls there are, the more bone fill may be expected.
  - Bone fill strongly depends on a plaque-free dentition (Fig. 11.**18**).

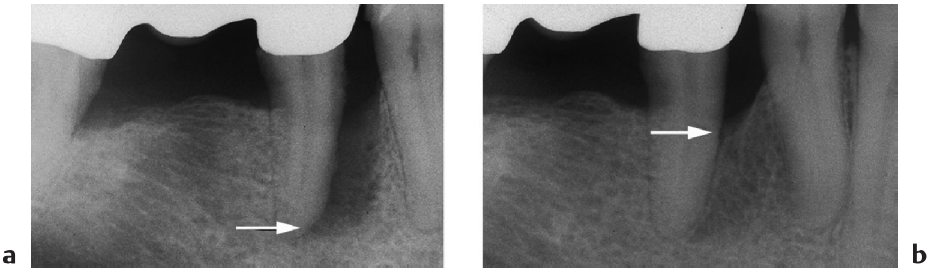


Fig. 11.**17** Bone fill in deep intrabony pockets.
a Three-wall bony pocket at second premolar, which extends up to the apex.
b About 70 % bone fill 12 months after flap surgery. Tooth remained vital. *Arrows* indicate the bottom of the bony lesion

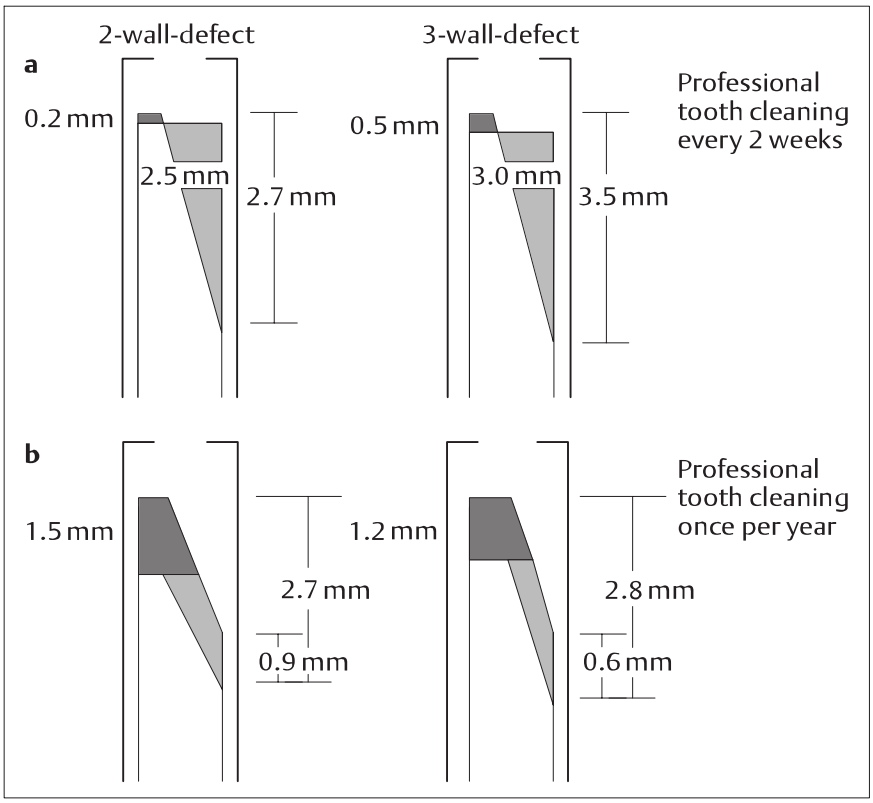


Fig. 11.**18** Bone fill after flap surgery may be expected mostly in two- and three-wall pockets and in plaque-free dentitions (Rosling et al. 1976).
a After 12 months only slight loss of crestal bone but large fill-in of the bony defect (■) was observed in individuals with extremely careful plaque control who underwent professional tooth cleaning every two weeks.
b In those who underwent yearly check-ups only, virtually no bone fill was observed, while crestal bone was resorbed (■)

Periodontal Wound Healing

General Remarks

- Until about 20 years ago, the main focus of periodontal therapy was control of the periodontal infection. The major aims of periodontal surgery were:
  - to provide *access* to the heavily colonized root surface;
  - to drastically change the *ecology* of the area in such a way as to inhibit bacterial recolonization.
- Periodontal lesions treated in this way usually heal with preservation of the anatomical defect caused by the inflammation. Clinical and radiological alterations include:
  - reduction of periodontal probing depth;
  - gain of clinical attachment;
  - filling in of bony defects.
- Whether true periodontal *regeneration* has occurred can only be assessed *histologically* after biopsy. This is generally not admissible in humans.
- Biological and functional regeneration means:
  - formation of new root cementum on a formerly bacterially colonized root surface and
  - principal fibers of a new periodontal ligament inserting in
  - newly formed alveolar bone.
- Important prerequisites for periodontal regeneration include:
  - presence of progenitor cells;
  - reestablishment of a biocompatible root surface;
  - exclusion of epithelium during wound healing;
  - stabilization of the wound area.

Presence of Progenitor Cells

- Periodontal regeneration requires specific cell activities of progenitor cells, which must proliferate, migrate into the wound area, differentiate, and eventually synthesize extracellular matrix components.
- Progenitor cells colonizing the periodontal defect should have specific capabilities to form cementum, bone and periodontal ligament fibers. They are assumed to reside:

- in the remaining periodontal ligament;
- in the adjacent alveolar bone;
- in blood.

Reestablishment of a Biocompatible Root Surface

- Exposed root surfaces exhibit certain *pathological alterations:*
  - Destruction of Sharpey's fibers anchoring in root cementum
  - Hypermineralization of cementum/dentin
  - Penetration of cementum and, in particular, dentin by toxins or even bacteria
- To make a contaminated root surface biologically acceptable for the organism again, certain modifications are necessary:
  - Scaling and root planing remove bacteria and toxins.
  - A thorough removal of the smear layer resulting after instrumentation is achieved by, e.g., citric acid, saturated tetracycline solution, or EDTA.

Exclusion of Epithelium

- Gingival epithelium is the periodontal tissue with the highest proliferation rate:
  - Proliferation starts in the initial phase of wound healing along the inner surface of the flap in an apical direction; after about one week the original bottom of the lesion is reached.
  - It adheres to the tooth surface by an epithelial attachment mechanism.
  - *Note:* Epithelium prevents contact between connective tissue and the root surface.
- The various connective tissues of the periodontium exhibit different regenerative potentials:
  - Contact between *gingival connective tissue* and the root surface results in *root resorption.*
  - Contact between *bone tissue* and the root surface leads to *ankylosis.*
  - Only *periodontal ligament* contains cells which can initiate cementogenesis and formation of desmodontal fibers.
- These observations, which mainly stem from animal experiments, led to the discovery of the biological principle of *guided tissue regeneration:*
  - The result of any wound healing depends on the kinds of cells populating the wound area.
  - For this reason, exclusion of epithelium may allow cells from the periodontal ligament to proliferate coronally onto the root surface.

Wound Stabilization

- *Stabilization of the wound area* is the most critical point during initial wound healing:
  - The blood clot beneath the flap is particularly sensitive to mechanical forces.
  - Disruption of the fibrin clot leads to apical proliferation of the epithelium.
- Wound healing of an incision is a precisely predictable process. Its early (hours) and medium-term (days) events are known in detail:
  - Wound healing commences with the immigration of chemotactically attracted cells, which start to clean the wound of any injured and necrotic tissue, foreign material, and microorganisms.
  - It ends with the production and maturation of extracellular matrix, which bridges the wound margins, supports cells and vessels, and restores resistance to functional strain.
  - Epithelial cells rapidly bridge the maturing fibrin clot.
- Three overlapping wound healing phases may be distinguished:
  - *Inflammation:*
    - Immigration of neutrophilic granulocytes which clean the wound area of bacteria and injured tissues.
    - Macrophages start repair.
  - *Formation of granulation tissue:*
    - Mediators of macrophages initiate angiogenesis and cell proliferation in the wound area, resulting in the formation of granulation tissue.
    - Proliferating cells migrate within the fibrin network and deposit a loose extracellular matrix of collagen, fibronectin, and proteoglycans.
    - The matrix contracts under the influence of cell–cell and cell–matrix contacts.
  - *Maturation and remodeling of granulation tissue:*
    - Increasing resistance to functional strain.

- This phase may last weeks or even months.

➤ *Note:* Wound healing of the periodontium is, in essence, much more complicated:
- The mucogingival flap is repositioned against a nonvascular, hypermineralized, firm root surface.
- This leads to tear-off of the fibrin clot, usually after a very short period of time.
- Subsequent apical proliferation of epithelium prevents regeneration of root cementum and periodontal ligament.

➤ In essence, two complementary concepts govern all therapeutic measures intended to promote periodontal regeneration:
- Prevention of apical proliferation of epithelium:
  - Historically, by repeat curettage during wound healing
  - Historically, by excision of gingiva with or without placement of a connective tissue graft
  - Nowadays, by using mechanical barriers
- Stabilization of the fibrin clot by:
  - Root conditioning with, e.g., citric acid, tetracycline, or EDTA
  - Coronal repositioning and fixation of the mucoperiosteal flap
  - Nonresorbable or resorbable membranes
  - Bone or bone substitutes (Table 11.**3**)
  - Tissue glues based on fibrin–fibronectin

Table 11.**3** Bone and bone replacement grafts

Human	• Autogenous grafts – Extraoral source – Intraoral source • Allogeneic grafts – Freeze-dried bone allografts – Demineralized freeze-dried bone
Bone substitutes	• Xenogeneic grafts – Bovine hydroxyapatite – Coral calcium carbonate • Alloplastic grafts – Polymers – Ceramics – β-Tricalcium phosphate – Hydroxyapatite – Bioactive glass

Bone and Bone Substitutes

➤ Bone replacement grafts may have various properties:
- *Osteogenesis:* new bone formation by osteoblasts contained in the transplanted graft itself.
- *Osteoinduction:* resident progenitor cells are induced to form new bone.
- *Osteoconduction:* bone formation by already committed cells close to the filling material, which is inert.

➤ *Autogenous bone* (autograft):
- If vital osteoblasts are transplanted, osteogenesis may be expected.
- No risk of transmission of infectious diseases.
- Disadvantage: second surgical site.

➤ *Allogeneic graft* (e.g., mineralized/demineralized freeze-dried bone allograft):
- Induces bone and cementum formation by differentiation factors: bone morphogenetic proteins (BMPs).
- *Warning:* Very small, persistent risk of HIV infection and Creutzfeld-Jacob disease.

➤ *Alloplastic graft* (e.g., tricalcium phosphate, hydroxyapatite, calcium carbonate, bioactive glasses):
- May be used as more or less inert filler for infrabony lesions. However:
  - Induction of bone or cementum formation has not been proven or is highly questionable.
  - Rather, proliferating cells from the periodontal ligament may be blocked.
  - Difficulties may arise in properly diagnosing persisting periodontal infections.

Growth and Differentiation Factors

➤ *Growth factors* are mitogenic polypeptides which locally or systemically influence the *growth* and *function* of different cells:
- *Autocrine effects:* growth-factor-secreting cells are themselves stimulated.
- *Paracrine effects:* the numbers of certain target cells, and thus of their products, are controlled.

➤ *Differentiation factors* control the *phenotype* of certain cells:
- Progenitor cells develop under the influence of differentiation factors into functioning, mature cells.

Table 11.**4** Activity of growth and differentiation factors; ++: greatly increased, +: increased, –: no or negative effect. (After Cochran and Wozney 1999)

	Fibroblast proliferation	Preosteoblast/ osteoblast proliferation	Extracellular matrix synthesis	Mesenchymal cell differentiation	Vascularization
Platelet-derived growth factor (PDGF)	++	++	–	–	+ (indirectly)
Insulin-like growth factor (IGF)	+	++	++	–	–
Bone morphogenetic proteins (BMPs)	–	+/–	+/–	++	++ (indirectly)
Transforming growth factor-β (TGF-β)	+/–	+/–	++	–	+ (indirectly)
Fibroblast growth factor (FGF)	++	++	–	–	–

  - For instance, in the presence of BMPs, mesenchymal precursor cells develop into osteoblasts.
- ➤ In the future, human recombinant growth and differentiation factors (Table 11.**4**) are expected to come into use for regeneration of periodontal lesions. The term "bioengineering" has been coined for this kind of therapy.
- ➤ At present, platelet-enriched plasma from the patient's own blood is employed in both periodontal and oral and maxillofacial surgery:
  - Enrichment is achieved using a cell separator.
  - High concentrations of platelet-derived growth factor (PDGF) and transforming growth factor-β (TGF-β) stimulate bone growth and periodontal ligament formation.

Root Surface Conditioning with Enamel Matrix Proteins

- ➤ During tooth development cells of Hertwig's epithelial root sheath secrete enamel matrix proteins (amelogenins):
  - This is necessary for the formation of acellular extrinsic fiber cementum
  - The therapeutic approach is to imitate these biological events during development as a means of stimulating periodontal regeneration.
- ➤ Enamel matrix proteins are derived from tooth germs of pigs (Emdogain):
  - Emdogain is pharmacologically safe, especially, it is not immunogenic.
  - After thorough root débridement during periodontal surgery and removal of the smear layer by EDTA, Emdogain is applied to the root surface.
- ➤ Both animal experiments and controlled clinical studies have produced very promising results:
  - Formation of both acellular extrinsic and cellular intrinsic fiber cementum.
  - Clinical attachment gain is more pronounced after Emdogain application than after conventional flap surgery and may be comparable to results after guided tissue regeneration (see below).
  - Long-term results emphasize the stability of the achieved gain of periodontal attachment and bone.

Guided Tissue Regeneration

Membranes

- Certain technical tricks are presently used in periodontal surgery to stabilize the wound area and exclude the epithelium during wound healing (Fig. 11.**19**):
  - so-called guided tissue regeneration, employing
  - non-resorbable and resorbable (bio-degradable) membranes (Tables 11.**5**, 11.**6**).
- First-generation membranes consist of expanded polytetrafluoroethylene (ePTFE, Gore-Tex periodontal material) and are still regarded as the standard for regenerative periodontal therapy by some practitioners:
  - They have to be removed in a second surgical operation after four to six weeks.
  - There is considerable risk of infection due to bacterial colonization of the membrane.
  - The membrane tends to collapse into the defect.
- Titanium-reinforced membranes facilitate better space-keeping and may even allow regeneration of horizontal bone loss.
- Resorbable membranes make a second operation for removal unnecessary. Clinical re-

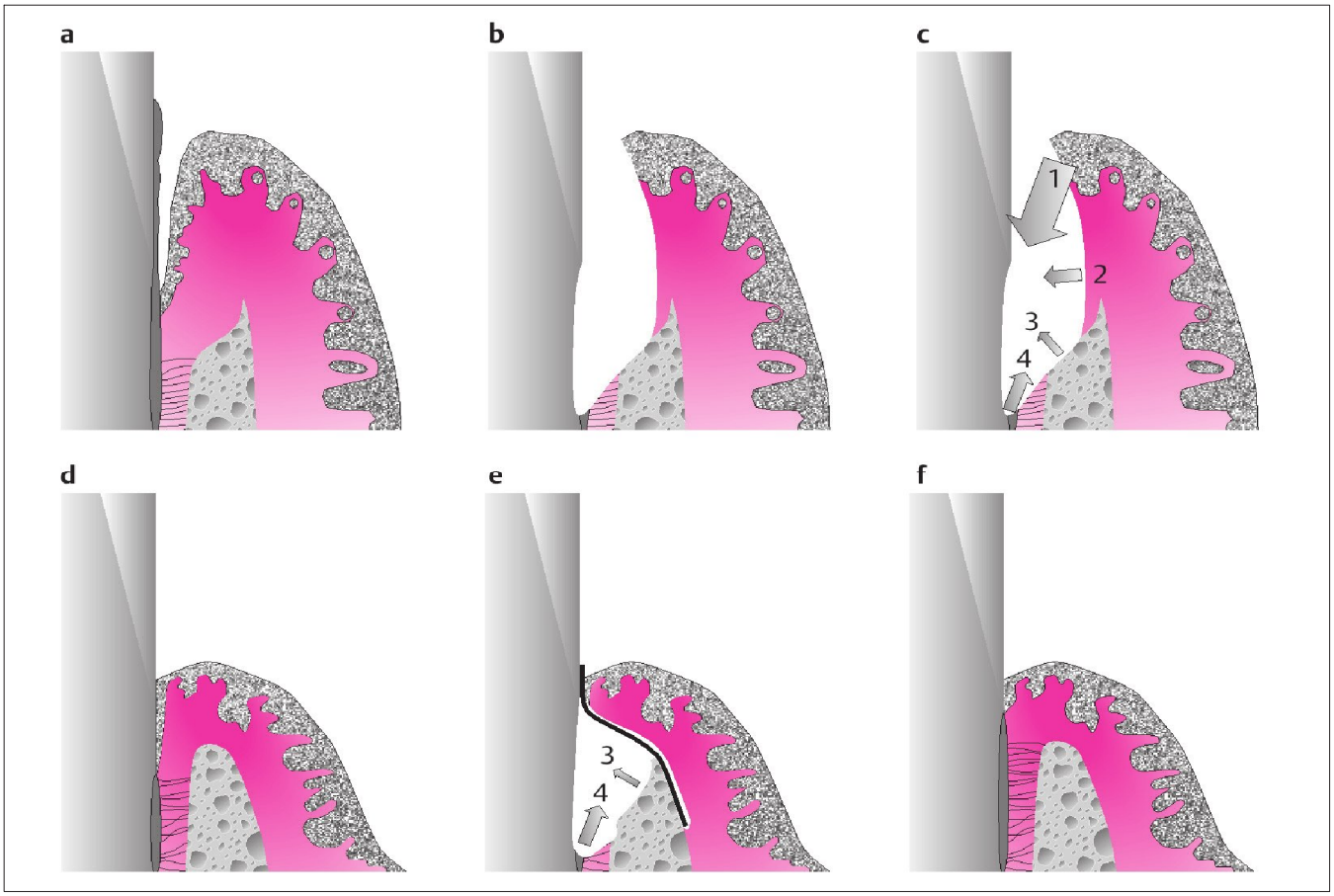


Fig. 11.**19** Current understanding of guided periodontal tissue regeneration.
a Preoperative situation with a bony pocket.
b After flap mobilization, granulation tissue is removed and root surface débrided.
c Epithelium (*1*) has the greatest proliferation rate, followed by gingival connective tissue (*2*).
d Therefore, undisturbed wound healing results in a long epithelial attachment, which protects the root surface from contact with cells of the periodontal ligament or bone. Nevertheless, at least in part, bone fill may take place.
e After placement of a mechanical barrier (membrane), cells of alveolar bone (*3*) and fibroblasts of the periodontal ligament (*4*) get a chance to colonize the wound area, which is simultaneously protected from tensile forces.
f This guided tissue regeneration may lead to the formation of new root cementum and functional fiber apparatus of a new periodontal ligament

Table 11.5 Requirements of optimum barriers for guided tissue regeneration

Requirements	Notes
Safety	Approval by responsible authorities (United States Food and Drug Administration, other national or international administration agencies)
Biocompatibility	The material must not be toxic; it must not trigger immunological or inflammatory reactions
Tight cervical occlusion	Sutures should be incorporated in the membrane
Adaptation to the alveolar bone	
Space-making ability	The membrane should not collapse into the defect; the membrane stiffness should adequately stabilize the wound area
Tissue integration	Connective tissue should be able to grow into the structured outer surface of the membrane
Permeability to tissue fluids and growth factors	At present speculative
Functional period	At least six weeks; longer may be advantageous
Easy handling	

Table 11.6 Some synthetic barriers for guided tissue regeneration

Material	Composition	Stiffness, space-making ability	Biologically degradable	Tissue integration	Notes
Gore-Tex Periodontal Material	Expanded polytetrafluoroethylene (ePTFE)	Quite good	No	Hardly any	Longest experience so far
Gore-Tex Periodontal Material, titanium-reinforced	ePTFE, titanium scaffold	Very good	No	Hardly any	For root coverage; may be tried for treatment of horizontal bone loss
Vicryl Periodontal Mesh	Polyglycolide/ polylactide (9:1)	No	Yes	Hardly any	Woven barrier which is resorbed within one to three months
Mempol	Polydioxanone	Good	Yes	Yes	Special structure on the outer surface is intended to allow tissue integration
Resolut Regenerative Material	Polyglycolide/ polylactide	Good	Yes	Yes	Resorption starts after four weeks and is complete within six months
Gore Osseoquest	Polyglycolide/ polylactide/ trimethylene carbonate	Good	Yes	Yes	Functional period longer than six months. Used especially for guided bone regeneration
Atrisorb	Poly-DL-lactide in a biocompatible vehicle of *N*-methyl-2-pyrrolidone	Good	Yes	Hardly any	Membrane is custom-made. The liquid phase sets under influence of tissue fluid. Good adaptation to the defect
Guidor Matrix Barrier	Poly-DL-lactide/poly-L-lactide, acetyltributyl citrate as softener	Good	Yes	Yes	Double-layered membrane with larger perforations on the outer surface for connective tissue integration, and smaller ones on the inner surface for permeation of tissue fluid

sults obtained with resorbable membranes have been very similar to those achieved with nonresorbable membranes. Natural products may be differentiated from synthetic material:
- Human collagen, e.g., lyophilized dura mater (allograft) or bovine collagen (xenograft) have certain disadvantages:
  - Poorly controlled, usually rapid, resorption.
  - Material may be immunogenic; sensitization against recipient's own collagen cannot be excluded.
  - (Very low) risk of disease transmission.
- Polyglycolide, polylactide, and copolymers of these aliphatic polyesters (Resolut regenerative material, Guidor matrix barrier, Vicryl mesh) have the following characteristics:
  - Biocompatible, safe materials
  - Special design facilitates stiffness of the membrane and allows tissue integration
  - Adequate functional period, in some cases up to 6 months.

Indications

- Guided tissue regeneration is intended to lead to considerable alteration of the local morphology of the periodontal defect. As a main result, the function and prognosis of the tooth should be improved.
- As regards to attachment gain, certain periodontal lesions respond considerably better to guided tissue regeneration than to conventional treatment modalities:
  - Two- and three-wall, at least 4-mm-deep, bony pockets
  - Buccal and lingual degree II furcation involvement of mandibular molars
  - Buccal degree II furcation involvement of maxillary molars
  - Bony defects after surgical removal of impacted wisdom teeth
  - Deep Miller class I or II gingival recessions

Contraindications

- General contraindications:
  - Systemic diseases that make operative risk unjustifiably high
  - Insufficient oral hygiene
  - Excessive smoking
- Specific contraindications relating to unfavorable defect morphology:
  - Horizontal bone loss and one-wall bony lesions
  - Various osseous lesions around multirooted teeth:
    - Degree I furcation involvement
    - Degree III furcation involvement
    - Degree II involvement of mesial and distal furcations of maxillary molars
    - Furcation involvement of premolars and wisdom teeth (there may be reasonable exceptions)
  - Miller class III or IV gingival recession
  - Teeth that are "hopelessly" periodontally diseased:
    - Lacking any remaining periodontal ligament for desired proliferation.
    - Excessive tooth mobility may interfere with wound healing.

Procedure

- The operating surgeon should strive for maximum soft tissue preservation for membrane coverage:
  - Disinfection.
  - Local anesthesia.
  - Incision:
    - Strictly intracrevicular.
    - Depending on the available interdental space, preparation of a modified (cf. Fig. 11.**21**) or simplified papilla preservation flap (cf. Fig. 11.**22**).
    - If needed, usually only one releasing incision about one tooth-width lateral to the defect is placed.
  - Mobilization of a full-thickness, mucoperiosteal flap; complete disclosure of the defect.
  - Removal of any granulation tissue.
  - Thorough scaling and root planing; if applicable, root conditioning with, e.g., citric acid, saturated tetracycline solution or EDTA.
  - Selection of an appropriate membrane, which is trimmed according to the bony defect. The membrane should completely cover the defect and overlap the bone margins by about 2 mm (Fig. 11.**20**).
  - It is tightly fixed to the neighboring teeth with sling sutures.
  - The flap is coronally advanced after careful dissection of the periosteum at its base.

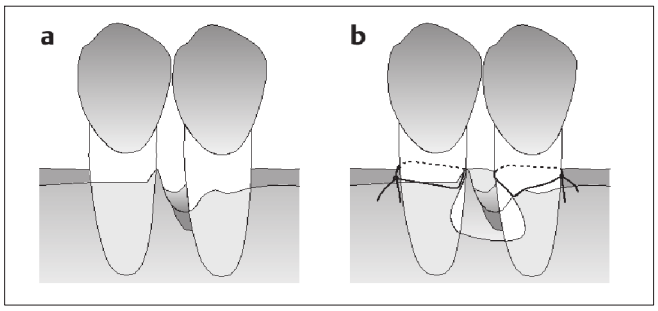


Fig. 11.**20** A trimmed membrane is placed over a combined one-, two-, and three-wall bony lesion and fixed to the neighboring teeth

- Primary closure of the interdental space and tension-free fixation of the flap over the membrane by using horizontal and vertical mattress sutures (Figs. 11.**21**, 11.**22**); note that sutures have to be left in place for an extended period.
- Additional interrupted sutures of finer suturing material (5-0) are used to approximate wound margins.

Postoperative Care

➤ The patient must be briefed about the delicate wound healing processes.

➤ Postoperative infection control:
- Mouth rinses twice daily with a 0.1–0.2% chlorhexidine solution for about four to six weeks. Toothbrushing must be avoided in the surgically treated area.
- During the first two weeks, wound healing should be checked every second or third day. If necessary, supragingival plaque at the gingival margin is carefully removed.
- If the membrane is exposed, the patient should apply a 1% chlorhexidine gel twice daily. *Note:* Secondary surgical coverage should never be performed.
- Antibiotics are not routinely prescribed.

➤ Suture removal after four to six weeks.

➤ If a nonresorbable membrane has been implanted, it is surgically removed after four to six weeks:
- Intracrevicular incision after local anesthesia.
- Mobilization of a small mucoperiosteal flap.
- Cutting of the sling suture.
- Removal of the membrane with tissue pliers.
- Careful removal of granulation tissue at the inner surface of the flap with a universal curette.
- Repositioning and fixation of the flap with interdental sutures.

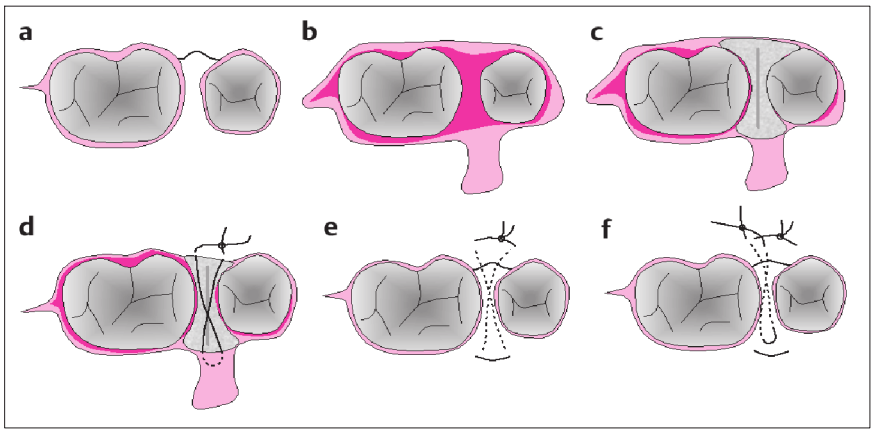


Fig. 11.**21** Modified papilla preservation technique (Cortellini et al. 1995).
a Semilunar incision to preserve the palatal papilla.
b Mobilization of the papilla.
c Titanium-reinforced membrane is trimmed and placed over the defect. Subsequently, the buccal periosteum is dissected at the base of the buccal flap to allow its tension-free advancement.
d A horizontal internal crossed mattress suture is first placed beneath the mucoperiosteal flap between the base of the palatal papilla and the buccal flap.
e, f The interproximal tissue can then be fixed, without any tension, with a second, vertical mattress suture

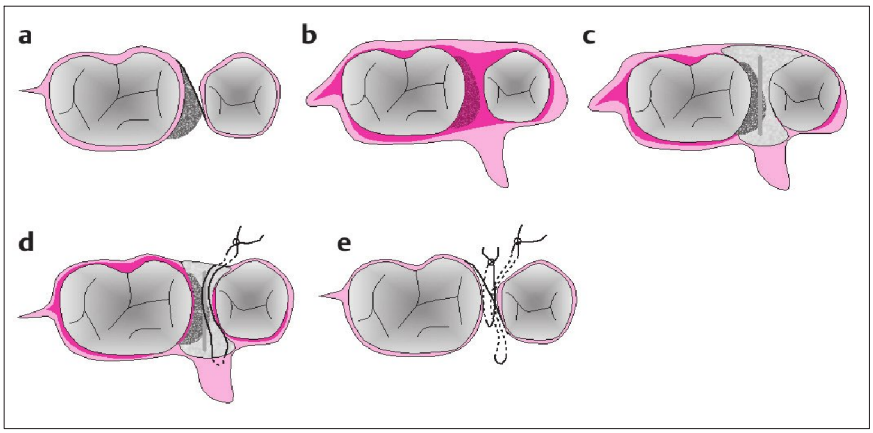


Fig. 11.**22** The simplified papilla preservation flap (Cortellini et al. 1999) is particularly recommended in cases where interdental space is narrow (e. g., at anterior teeth) and where there is an intrabony lesion around only one tooth.

a Oblique incision starts at the buccal line angle of the involved tooth to reach the midinterproximal portion of the papilla of the adjacent tooth.

b Elevation of a full-thickness flap.

c Titanium-reinforced membrane is trimmed and placed over the defect. The buccal periosteum is then dissected at the base of the buccal flap to allow its tension-free advancement.

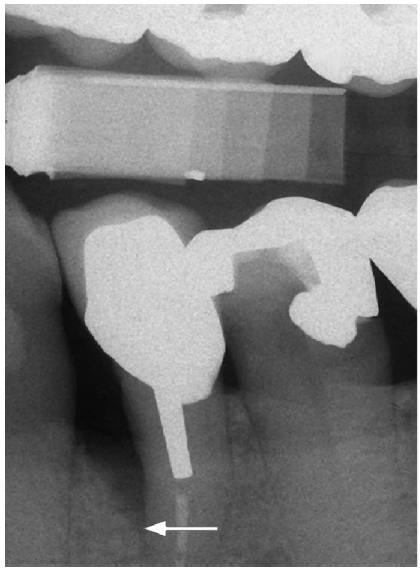
d An offset internal horizontal mattress suture is positioned running from the base of the keratinized tissue at the midbuccal aspect of the noninvolved tooth to a symmetrical location at the base of the lingual/palatal flap. The suture rubs against the interproximal root surface, hangs on the residual interproximal bone crest, and is anchored to the lingual/palatal flap.

e Primary closure is obtained by one or two interrupted sutures or vertical mattress suture, depending on available space

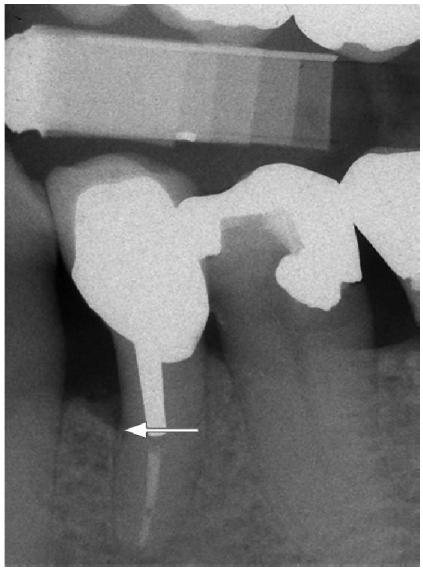
– If complete coverage is not possible, placement of a free gingival graft should be considered to protect the regenerated tissue.

Critical Assessment

➤ The dentist should take care not to foster unrealistically high expectations on the part of the patient:



a



b

Fig. 11.**23**

a Three-wall bony lesion at lower first premolar before regenerative treatment and

b after three years. Note about 60 % bone fill. *Arrows* indicate the bottom of the defect

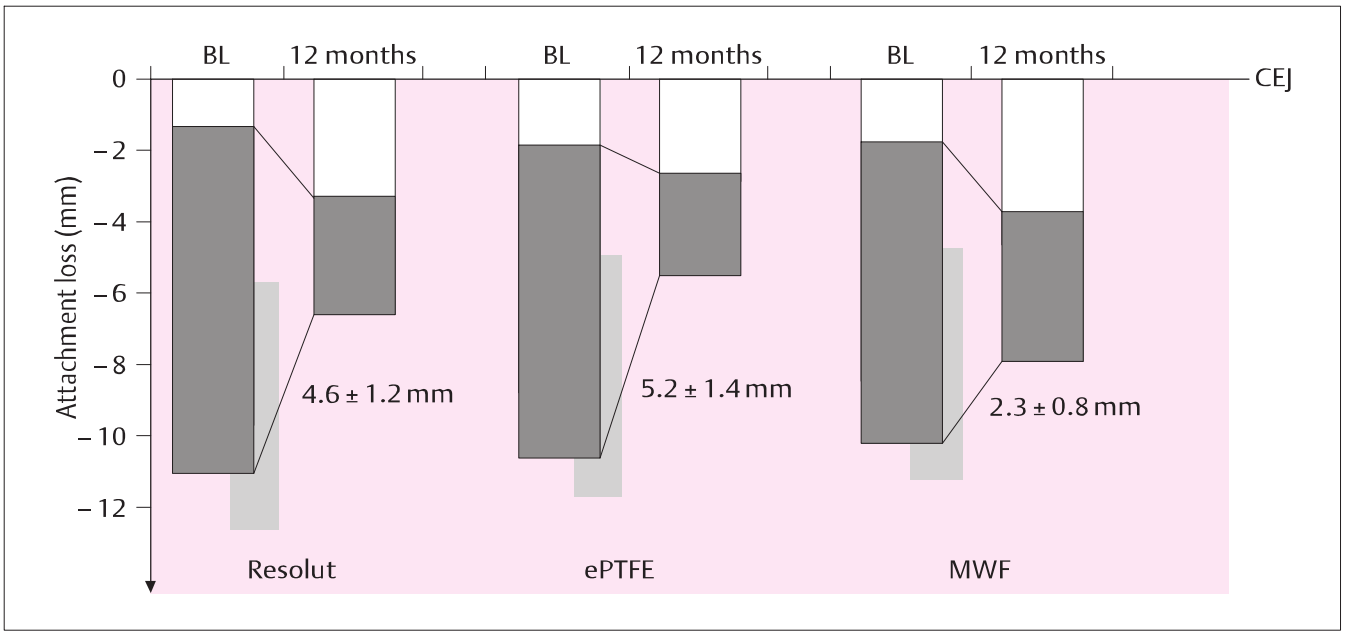


Fig. 11.**24** Controlled clinical trial (Cortellini et al. 1996) comparing postoperative results after 12 months in bony pockets after using resorbable (Resolut) and nonresorbable membranes (ePTFE) for guided tissue regeneration and conventional access flap operations (modified Widman flap). Excellent comparable attachment gain of almost or about 5 mm was observed after guided tissue regeneration, particularly in bony pockets (▒). Considerably less favorable results of slightly more than 2 mm attachment gain were calculated, on average, after conventional flap surgery. ePTFE membranes resulted in somewhat less recession (□). ■ periodontal probing depth

- Guided tissue regeneration is generally not intended to be a treatment that will save "hopeless" teeth.
- At present, the place of this treatment in daily practice may be limited:
  - The technique is quite complicated.
  - Postoperative results may be not predictable
  - The range of indications is limited.
- From a biological point of view, wound healing processes are initiated, mostly reparative, that are not really predictable:
  - Cellular intrinsic fiber cementum without functional importance is formed.
  - Moreover, new cementum may insufficiently be attached to the root surface.
- Nevertheless, favorable results have been reported in numerous case reports (Fig. 11.**23**) and clinical investigations (Fig. 11.**24**).

➤ A recent metaanalysis of randomized controlled clinical trials came to the following conclusions:

- Guided periodontal tissue regeneration was more effective than open flap débridement in improving clinical attachment levels.
- However, there is marked variability between studies.
- This variability limits the general conclusions that can be drawn. Thus, evidence of a consistent and clinically relevant benefit has to be questioned.

➤ Guided tissue regeneration may successfully be combined with autogenous bone grafts or bone allografts.

Treatment of Furcation-Involved Teeth

General Remarks

➤ Because of the difficulty of access and the bizarre microrelief of the furcation area, treatment of furcation-involved teeth has always been rather problematic. *Note:* Furcation in-

volvement drastically impairs the prognosis of a multirooted tooth.

- Dependent on tooth type, degree of furcation involvement, and several general factors such as course/expression of periodontal disease, patient's age and condition of health, compliance, etc., *different treatment modalities* are indicated:
  - *Conservative treatment*, i. e., mechanical instrumentation of bacterially colonized root surfaces with or without osteoplasty
  - *Resective procedures* such as root amputation, hemisection, premolarization, tunneling
  - *Periodontal regeneration*

Fundamental Morphological Terms

- *Anatomic root complex* (Fig. 11.**25**): that part of a tooth located apical to the cementoenamel junction.
- *Root cone:* a constant morphological unit. At a given level, a root is classified as either nonseparated, i. e., connected to one or more other root cones, or separated, i. e., separated from one or more other root cones.
- *Root component* (relates only to molars):
  - The root complex of maxillary molars consists of three root components.
  - The root complex of mandibular molars is composed of two root components.
  - Each root component is composed of two or a maximum of three root cones.
- *Separation structures:*
  - *Furcation:* complete separation between root cones or roots.
  - *Root groove:* incomplete separation between root cones or roots.
- *Root trunk:* the part of the root complex located coronal to the furcation.
- *Furcation entrance:* transitional area between the mainly vertical and the mainly horizontal part of the furcation.
- *Fornix:* roof of the furcation.
- *Degree of separation* between two root cones or roots: the maximum furcoapical extent in relation to the maximum cervicoapical extent of the root complex.
- *Degree of divergence:* the angle formed by the cervical half of the long axes of two root cones or roots.

Structures Within Furcations

- As a product of separated enamel organs derived from Hertwig's epithelial root sheath, paraplastic enamel formation may frequently be observed in the area of the furcation entrance, which promotes bacterial proliferation and should therefore be removed:
  - *Enamel projections* may be classified according to length:

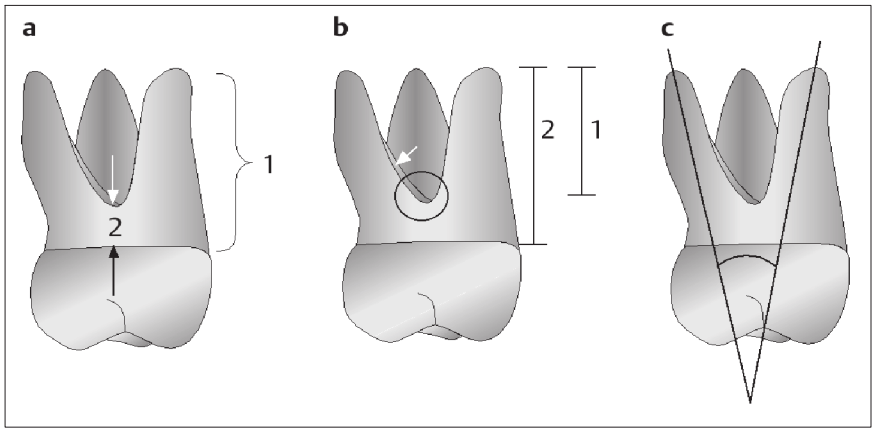


Fig. 11.**25** Anatomic root complex.
a The root complex (*1*) is that part of the tooth which is located apical to the cementoenamel junction. The root trunk (*2*) is the part of the root complex, which extends from the cementoenamel junction to the furcation entrance.
b Degree of separation: the maximum furcoapical extension (*1*) in relation to the maximum cervicoapical extension (*2*) of the root complex. Complete separation of the roots (○) by furcation; incomplete separation (*arrow*) by a root groove.
c Degree of divergence

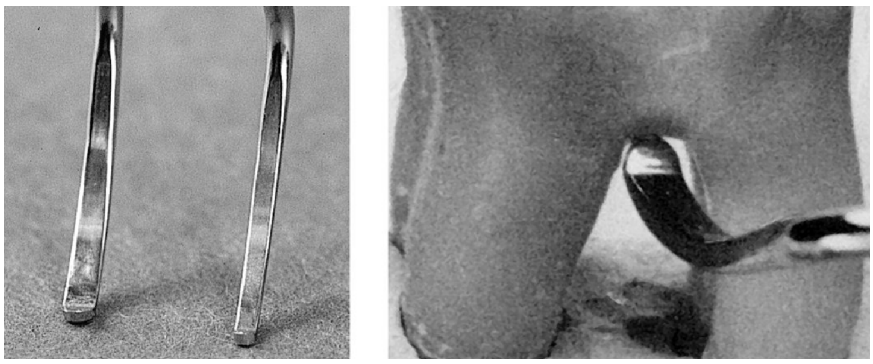


Fig. 11.**26** **a** Special furcation curettes, 1.3 mm and 0.9 mm wide, developed by Quétin for thorough instrumentation of the fornix of the furcation (**b**)

  - Class I: small extensions not extending beyond the root trunk
  - Class II: medium-sized spurs extending up to the furcation entrance
  - Class III: lancet-like projections extending into the furcation
- Enamel islands, drops, or pearls

➤ There is a *marked formation of cellular mixed stratified cementum* in the furcation area:
- Variably high ridge on two-rooted premolars
- Central, mesiodistal cementum bulge on mandibular molars
- T-shaped bulge on maxillary molars.

➤ The bizarre microrelief promotes bacterial colonization:
- Depressions and niches
- Accessory pulp channels
- Blind openings

Conservative Furcation Therapy

➤ Instrumentation of root surfaces is mainly performed during flap operations. The following instruments may be used:
- Curettes, e.g., Columbia 4R/4L, Langer SL17/18; special furcation curettes SQMD1, SQBL1 (Fig. 11.**26**)
- In certain cases Hirschfeld periodontal files
- Fine (40 µm) and extra-fine (15 µm) diamond-coated rotating burs
- Compressed air-driven sonic scalers

➤ Indication: degree I or incipient degree II furcation involvement. Odontoplasty in cases of enamel projections or pearls.

➤ *Note:* If there is advanced furcation involvement, conservative measures may only result in a transient improvement of the situation:
- In particular horizontal attachment gain should not be expected after conventional (conservative) treatment because of:
  - poor access to the furcation area;
  - the possibility of rapid bacterial recolonization.
- Therefore, frequent follow-up and, if required, retreatment are mandatory.

Root Amputation and Hemisection

➤ Resective measures are mainly performed to preserve strategically important teeth.

➤ The range of *indications is* wide and not confined to periodontal disease:
- *Periodontal:*
  - Degree II and III furcation involvement, in particular of first and second molars.
- *Endodontal:*
  - Obstructed root canal inaccessible to instrumentation
  - Post and core which cannot be removed
- *Iatrogenic:*
  - Broken endodontic instrument which cannot be removed from the root canal
  - Class IV root perforation, i.e., perforation into the furcation area
  - Class II root perforation, i.e., intraalveolar perforation within the middle root third
- *Other reasons:*
  - Intractable root or furcation caries
  - Rarely, as fractional extraction during orthodontic treatment.

- Definitions:
  - *Root amputation:* removal of one or several roots whilst preserving most parts of the crown; mainly relates to maxillary molars.
  - *Hemisection:* removal of a root together with the corresponding crown portion; generally the case for mandibular molars.
- Careful treatment *planning* is mandatory. Resective procedures should never be conducted ad hoc:
  - Appropriate endodontic treatment should be performed long enough in advance before the planned resection; in general, a temporary root canal filling with, e.g., $Ca(OH)_2$ is sufficient:
    - To facilitate the differential diagnosis in cases of toothache
    - To become aware of possible complications during endodontic treatment
  - Vital amputation should be avoided in any case:
    - Leads inevitably to chronic or even acute pulpitis
    - Full-scale root canal treatment, which may be necessary after some time, might become impossible.
- Procedure for root amputation (Fig. 11.**27**):
  - Disinfection.
  - Local anesthesia.
  - Mobilization of a mucoperiosteal flap to disclose the whole furcation area.
  - Removal of all granulation tissue.
  - Separation of the root with a needle-shaped diamond-coated bur:
    - Continuous cooling with sterile, physiological saline is mandatory.
    - *Warning:* beware damaging the root complex that is to be kept, and neighboring teeth.
    - Definitive separation of the root from the remaining root complex with a periosteal or thin luxating elevator; the patient

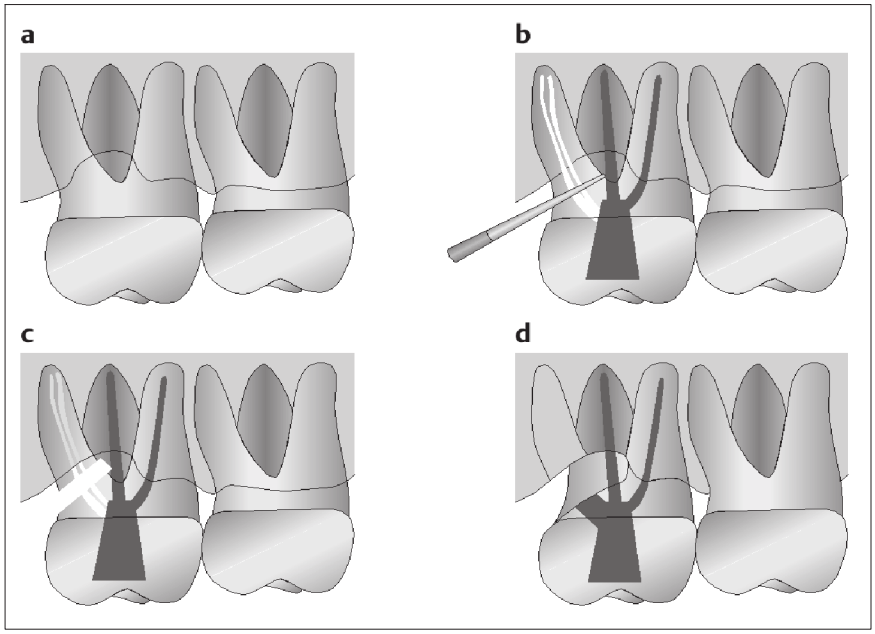


Fig. 11.**27** Root amputation.

a Advanced buccal–mesial furcation involvement of a maxillary first molar. The mesial root is to be removed.

b, c Definitive endodontic treatment of the distal and palatal root canal, temporary filling with calcium hydroxide in both mesial canals. After flap mobilization and removal of the granulation tissue, the mesial root is separated with a needle-shaped diamond bur from the remaining tooth. Cooling with sterile physiological saline is mandatory.

d After careful osteotomy the root can easily be removed with a small luxating elevator or root pliers. The amputation area has to be smoothened, and the opened root canal obturated with a small composite filling (may be done after wound healing)

must be prepared in advance for the cracking sound.
- Removal of the root with luxating elevator or root pliers.
- If required, osteoplasty with Schluger's bone file or universal curette.
- Smoothing of the amputation area with fine (40 µm) and extra-fine (15 µm) diamond-coated burs; continuous cooling is mandatory.
- Scaling and root planing.
- Repositioning of the flap; if necessary, advancement of the flap after dissecting the periosteum at its base.
- Approximation of the flap margins and, if possible, tight closure with interrupted sutures.
- In all cases, placement of a periodontal dressing (CoePak).

➤ Preparation of a small cavity and placement of a composite restoration to obturate the opened root canal; this may be done after wound healing.

➤ Usual postoperative infection control (mouth rinses with 0.1–0.2 % chlorhexidine solution) and care.

➤ Restorative treatment:
- Depending on the amount and shape of the remaining tooth substance, in most cases placement of a crown or partial crown is essential.
- Emerging problems with the morphology of the tooth remnants must be considered. If the tooth is caries-free, a small occlusal restoration may be placed.

➤ Procedure for hemisection of mandibular molars (Fig. 11.**28**):
- Disinfection.
- Local anesthesia.
- Immediately before flap mobilization, the crown portion of the tooth should be separated as far as the floor of the pulp chamber.
- Generous preparation at the expense of the root to be extracted; the cutting direction may be given by a straight periodontal probe inserted into the furcation.
- Mobilization of a mucoperiosteal flap.

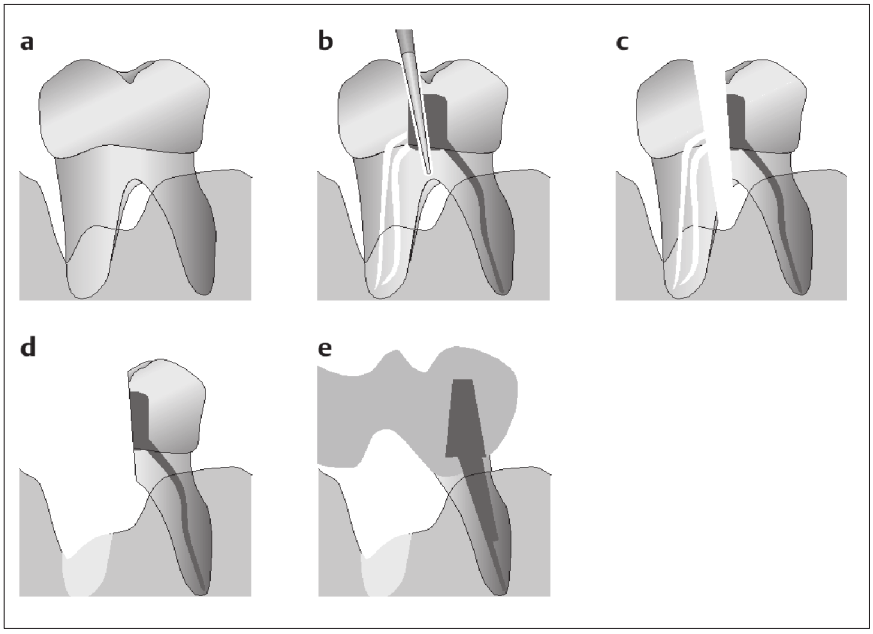


Fig. 11.**28** Hemisection of a mandibular molar.
a Advanced furcation involvement of a mandibular first molar. The mesial root is to be removed.
b Root canal therapy. Temporary root canal filling of the mesial canals with calcium hydroxide. Before mobilization of a mucoperiosteal flap, the crown portion of the tooth has to be separated as far as the floor of the pulp chamber.
c Final separation of the tooth (at the expense of the part to be extracted).
d The root is removed with root pliers. **e** A fixed partial denture is incorporated

- Further separation of the tooth with a needle-shaped diamond-coated bur:
  - Continuous and sufficient cooling with sterile physiological saline
  - Definitive separation of the root from the remainder of the tooth with a periosteal or small luxation elevator
- Extraction of this part of the tooth with root pliers.
- Removal of all granulation tissue; thorough scaling and root planing.
- Smoothing of the cut tooth surface with fine (40 µm) and extra-fine (15 µm) diamond-coated burs; continuous cooling is mandatory.
- Interrupted sutures.
- Periodontal pack (CoePak).

➤ Usual postoperative infection control (mouth rinses with 0.1–0.2 % chlorhexidine solution) and care.

➤ Restorative treatment after about three to four months:
- Placement of post and core.
- Incorporation of a fixed partial denture with the hemisectioned tooth as abutment. In some cases, the remainder of the tooth may serve as abutment for a single crown.

➤ Critical assessment:
- Resective measures do not improve the prognosis of the tooth per se.
- Technical problems especially that emerge during restorative treatment may affect the long-term prognosis of root-resected teeth:
  - Errors during endodontic treatment with immediate loss of the tooth, e.g., root fracture or perforation.
  - Endodontic errors that carry a risk of late complications, e.g., insufficient instrumentation, insufficient quality of root canal filling.
  - Loss of retention of the restoration.

➤ *Note:* The individual risks of every treatment are multiplied together to give the total failure risk, which thus increases greatly with the number of treatment steps. Long-term tooth preservation can only be expected if the entire treatment has been performed correctly.

Premolarization

➤ *Premolarization* is only performed—and then rarely—in mandibular molars:
- After successful root canal treatment the tooth is separated with a needle-shaped diamond-coated bur.
- If required, post and core are placed; thereafter single crowns are incorporated on both roots.
- If the tooth is the most distal and there is only a narrow space between the roots, orthodontic distalizing and incorporation of a small fixed partial denture may be possible.

➤ Indications:
- Degree II or III furcation involvement of mandibular first molars in which proximal bone has largely been preserved.
- Important prerequisites: divergence more than 30° and separation three-quarters or more.

Tunnel Preparation

➤ Opening and exposure of the furcation entrance to enable daily tooth brushing of the area. Prerequisites:
- Excellent oral hygiene.
- Low caries susceptibility; in some cases oral load of mutans streptococci and lactobacilli as well as salivary flow rate and buffering capacity should be assessed.
- Periodical recall, excellent compliance.

➤ Indications:
- Advanced degree II or degree III furcation involvement of mandibular first molars.
- Divergence more than 30° and separation more than three-quarters.

➤ Contraindications:
- Low divergence and separation
- High caries susceptibility. *Warning:* Beware of root caries in the furcation area
- Doubt about compliance of the patient.

➤ Procedure for tunnel preparation of mandibular molars (Fig. 11.**29**):
- Disinfection.
- Local anesthesia.
- Incisions:
  - Intracrevicular incision on the buccal aspect.
  - Usually, two vertical releasing incisions bound the surgical site mesial and distal

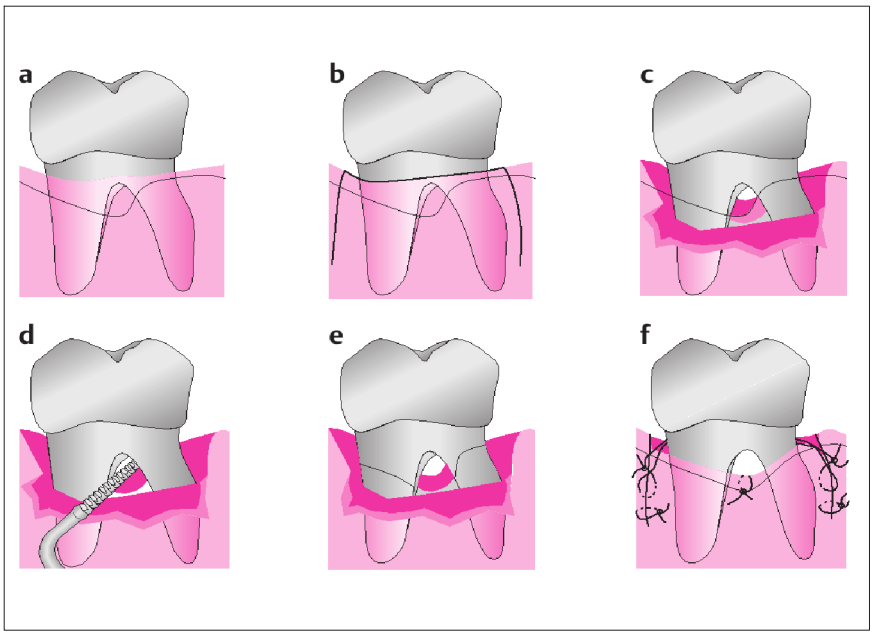


Fig. 11.**29** Tunnel preparation on a mandibular molar.
a Through-and-through furcation in a mandibular first molar. Proximal bone is largely preserved.
b Intracrevicular incision, vertical releasing incisions laterally.
c Mucoperiosteal flap is mobilized and granulation tissue removed.
d Osteoplasty with a Sugarman bone file (cf. Fig. 11.**30b**).
e Following widening of the furcation area, all desmodontal fibers and root cementum must be removed to prevent reattachment.
f Suturing: periosteal sutures (cf. Fig. 11.**31**) for apical repositioning of the flap, interradicular suture, interrupted sutures for releasing incisions

to the treated tooth at a distance of about half of a tooth's width.
- Lingually, the incision is performed about 0.5 mm paramarginally.

– Mobilization of a mucoperiosteal flap:
  - Buccally, beyond the mucogingival border.
  - Lingually, up to the bone crest.

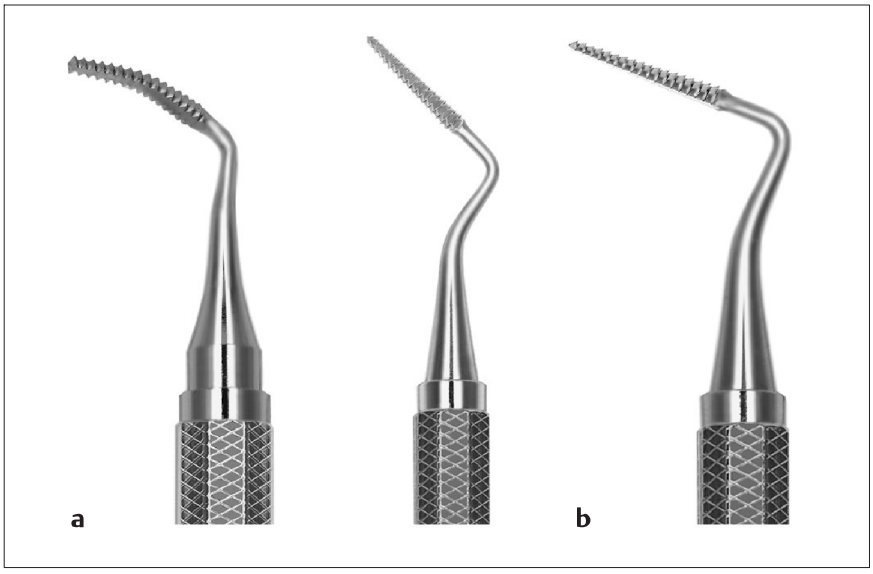


Fig. 11.**30** Bone files:
(**a**) Schluger, (**b**) Sugarman

- Removal of all granulation tissue.
- Careful ostectomy within the furcation using bone files (Fig. 11.**30**):
- Smaller Sugarman files are used in the beginning, followed by larger Schluger files.
- Bone may also be removed by bud-shaped burs in a reducing hand piece; sufficient cooling with sterile physiological saline is mandatory.

– Scaling and root planing:
- Removal of all bacterial deposits.
- Complete removal of all supraalveolar desmodontal fibers and root cementum to prevent reattachment of connective tissue.

– Lingually, the flap is shortened to expose the furcation.
– The buccal flap is apically repositioned and secured with periosteal sutures (Fig. 11.**31**). Interrupted sutures are used for releasing incisions.
– A periodontal dressing (CoePak) is applied:
- to secure the flap position;
- to keep the furcation area open.

➤ Usual postoperative infection control (mouth rinses with 0.1–0.2 % chlorhexidine solution) and care.
➤ Critical assessment:
– Tunnel preparation alters the unfavorable morphology of the furcation area, enabling the patient to clean the furcation on a daily basis.
– A major disadvantage is a frequently occurring pulpitis-like sensation:
- Since cementum should be completely removed from the entire furcation area, the dentinal tubules are opened over a wide area.
- In addition, accessory channels may connect the furcation with the pulp chamber.

– Dentinal hypersensitivity interferes with patient compliance; adverse effects are:
- High risk of new soft tissue closure requiring repeat surgical intervention
- Extended root caries in the furcation area; this is usually an indication for immediate extraction.

– Alternating application of fluoride (in the evening) and 1 % chlorhexidine gel (in the morning) with appropriate brushes is highly recommended (Fig. 11.**32**). In refractory cases the tooth might be devitalized.

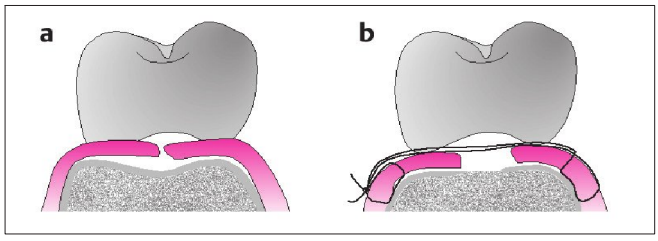


Fig. 11.**31** An apically repositioned mucoperiosteal flap with ostectomy is planned (**a**). Fixation of the buccal and lingual flaps in an apical position with periosteal suture (**b**)

Regenerative Procedures

➤ Indications:
– Mandibular molars with moderately advanced (degree II) buccal and/or lingual furcation involvement.
– In some cases, maxillary molars with a buccal degree II furcation involvement.

➤ Contraindications:
– General:
- Severe systemic disease interfering with surgical procedures
- Poor patient compliance
- Excessive smoking

Fig. 11.**32** In order to keep the furcation open, to remove any bacterial plaque, and to apply fluoride and chlorhexidine gels, a molar with a tunnel must be cleaned with interproximal brushes on a daily basis, if necessary from both the buccal and the lingual entrance

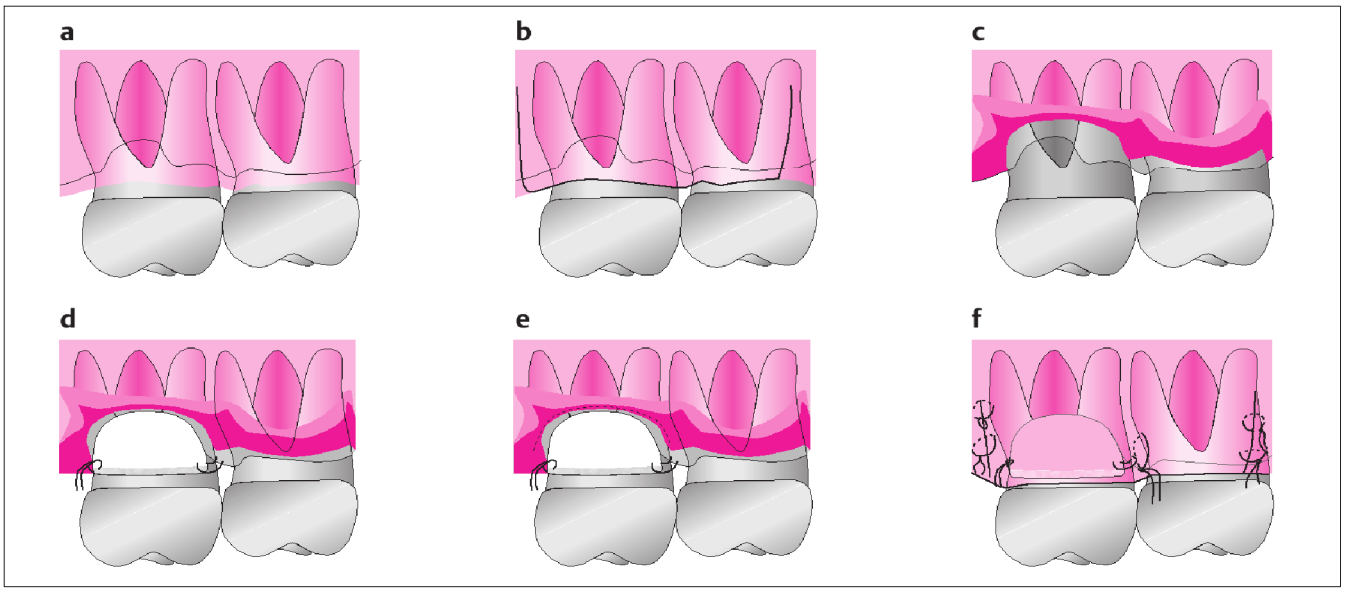


Fig. 11.**33** Regenerative furcation therapy.
a Buccal degree II furcation involvement of a maxillary first molar. Favorable situation for guided tissue regeneration: no proximal bone loss, mesial and distal furcations are not affected.
b Intracrevicular incision; two vertical releasing incisions are made laterally.
c A mucoperiosteal flap is mobilized and the granulation tissue removed, with thorough scaling and root planing.
d A suitable membrane is trimmed and fixed to the tooth with a sling suture; the membrane should overlap the bone margin by about 2–3 mm.
e Dissection of the periosteum at the base of the flap and sharp preparation of a split flap for flap advancement.
f The flap can now be coronally displaced without any tension. Tight suturing with vertical mattress suture and fine interrupted sutures to further approximate the wound margins in the proximal region; interrupted sutures for vertical incisions

- Local:
  - Degree I furcation involvement, except in combination with a deep intrabony pocket
  - Through-and-through (degree III) involvement
  - Complicated furcation involvement in maxillary molars without any chance of sufficient débridement, e.g., degree II involvement of a distopalatal furcation
  - Combinations with advanced proximal bone loss

➤ Procedure (Fig. 11.**33**):
- Disinfection.
- Local anesthesia.
- Incisions should be made with a view to preserving all soft tissue as far as possible:
  - Buccal and lingual/palatal intracrevicular incisions.
  - Two vertical releasing incisions bound the surgical site at a distance of about half of a tooth's width mesial and distal to the treated tooth.
- Mobilization of the full flap beyond the mucogingival border.
- Complete removal of all granulation tissue:
  - Disclosure of the entire furcation defect and surrounding bony pockets
  - If the defect morphology is unfavorable (communicating furcations, proximal bone loss extending to a level apical to the furcation entrance, deep bony pockets), alternative treatment modalities must be chosen: tunnel preparation or hemisection, in certain cases even extraction.
- Scaling and root planing:
  - The critical step, which basically determines the success or failure of the operation. *Note:* Special operative tricks are meaningless unless the infection can be controlled.

- If necessary, the furcation entrance should be widened (osteoplasty) to gain access to the furcal root surfaces.
- The fornix of the furcation is débrided with universal curettes, e.g., Columbia 4R/4L, Langer curette SL17/18; special furcation curettes SQBL1, SQMD1 (see Fig. 11.**26**).
- Sonic scalers in combination with extra-fine diamond-coated burs (15 µm) may be used.

– Selection and trimming of the membrane:
  - The membrane should overlap the bone margins by about 2–3 mm.
  - It is tightly fixed with a sling suture; the knot should be placed interproximally.
– The mucoperiosteal flap should cover the membrane completely without any tension:
  - Dissection of the periosteum at the base of the flap.
  - Further sharp preparation of a split flap.
  - This allows tension-free coronal advancement of the flap.
– Tight suturing of the mucoperiosteal flap (Figs. 11.**11**, 11.**33**):
  - Interdentally, a vertical mattress suture is placed.
  - Additional interrupted sutures for further wound margin approximation.
  - Interrupted sutures for vertical releasing incisions.
– No periodontal dressing should be applied.

➤ Postoperative care:
– Antibiotics are not routinely prescribed
– Postoperative follow-up sessions every second or third day for the first two weeks.
– If necessary, supragingival bacterial deposits in the surgical area are carefully removed.
– If the membrane is exposed, no attempt at secondary surgical covering should be made.
– Postoperative infection control:
  - No toothbrushing in the surgical site for about four to six weeks.
  - Chemical plaque control: mouth rinses with 0.1–0.2 % chlorhexidine solution twice daily.
  - If the membrane is exposed after some days or weeks, the patient should apply 1 % chlorhexidine gel with a cotton swab twice daily.
– A nonresorbable membrane must be surgically removed after four to six weeks.

➤ Critical assessment:
– Guided tissue regeneration can markedly improve the prognosis of furcation-involved teeth if the indications are strictly observed (Fig. 11.**34**).
– In comparative clinical studies, better healing results were observed in the following situations:
  - Buccal and/or lingual degree II involvement of mandibular molars.

a

b

Fig. 11.**34**
a Degree II furcation involvement and three-wall intrabony pocket at a lower first molar. Regenerative treatment was accomplished with a synthetic, biologically degradable membrane.
b Considerable bone fill in the furcation and the intrabony pocket 12 months after therapy

- Isolated buccal degree II involvement of maxillary molars.
- Best results can be expected in keyhole-like furcations with a small furcation area.
- Comparable results are obtained with resorbable and nonresorbable membranes.
- Exclusive or additional use of bone substitutes (e. g., demineralized freeze-dried bone allografts), or root conditioning with, e. g., citric acid, did not lead to superior results.

– *Note:* Conversion of moderately advanced furcation involvement (degree II) into slight (degree I) involvement may be regarded a success. Slight involvement can then be managed in the context of supportive care.

Possible Treatment Strategies

➤ The decision for one treatment modality or another is essentially influenced by the following factors (Fig. 11.**35**):
– Tooth type and site of involvement
– Degree of furcation involvement
– Strategic importance of the tooth
– Patient's willingness and ability to cooperate
– The question as to whether extensive restorative treatment is planned

➤ In individual cases several further factors have to be considered:
– General:
- Patient's age and health
- Form or expression of periodontitis
- Declared wishes of the patient

– Local:
- Amount of remaining periodontal supportive apparatus at individual roots
- Mobility of individual roots (cannot be determined preoperatively)
- Anatomic–topographic relationship between roots
- Caries and endodontic situation

➤ An effective treatment of furcation-involved *premolars* is to a large extent limited:
– The root complex usually exhibits only a small degree of separation.
– Very conical roots, resulting often in considerably increased tooth mobility.
– Maxillary premolars, on the other hand, are usually of high strategic importance:
- Palliative measures are often possible: repeat scaling and root planing.
- However, it may be difficult to decide when the time has come to extract.

➤ Furcation-involved *wisdom teeth* are usually of low strategic importance and can be extracted in an early phase of the treatment (there may be reasonable exceptions to this).

Mucogingival Surgery

General Remarks

➤ Functional or esthetic mucogingival disorders include in particular:
– Aberrant (i. e., marginal insertion of) lip, cheek, or tongue frenula
– Decreased vestibular depth
– Localized or generalized gingival recession

➤ Mucogingival surgery in the strict sense refers to certain operative techniques for plastic surgical correction of tooth-surrounding soft tissue, including:
– morphology,
– position,
– amount of tissue.

➤ The aim of *plastic periodontal surgery* is the correction of anatomical, developmental, traumatic, or disease-derived defects of gingiva, alveolar mucosa, and alveolar bone:
– Widening of the zone of attached and keratinized gingiva
– Correction of soft tissue defects of the mucogingival region
– Correction of defects of the alveolar process
– Pocket elimination procedures preserving the amount of keratinized gingiva.

Widening of the Zone of Keratinized Tissue with a Free Gingival Graft

➤ There are great inter- and intraindividual differences in gingival width:
– Minimal width of less than 3 mm on average is found at the vestibular aspect of canines and premolars of the mandible.
– A narrow band or even absence of attached gingiva may be a normal variant and/or due to gingival recession.
– *Note:* If oral hygiene is optimal, the gingival margin may be kept inflammation-free and

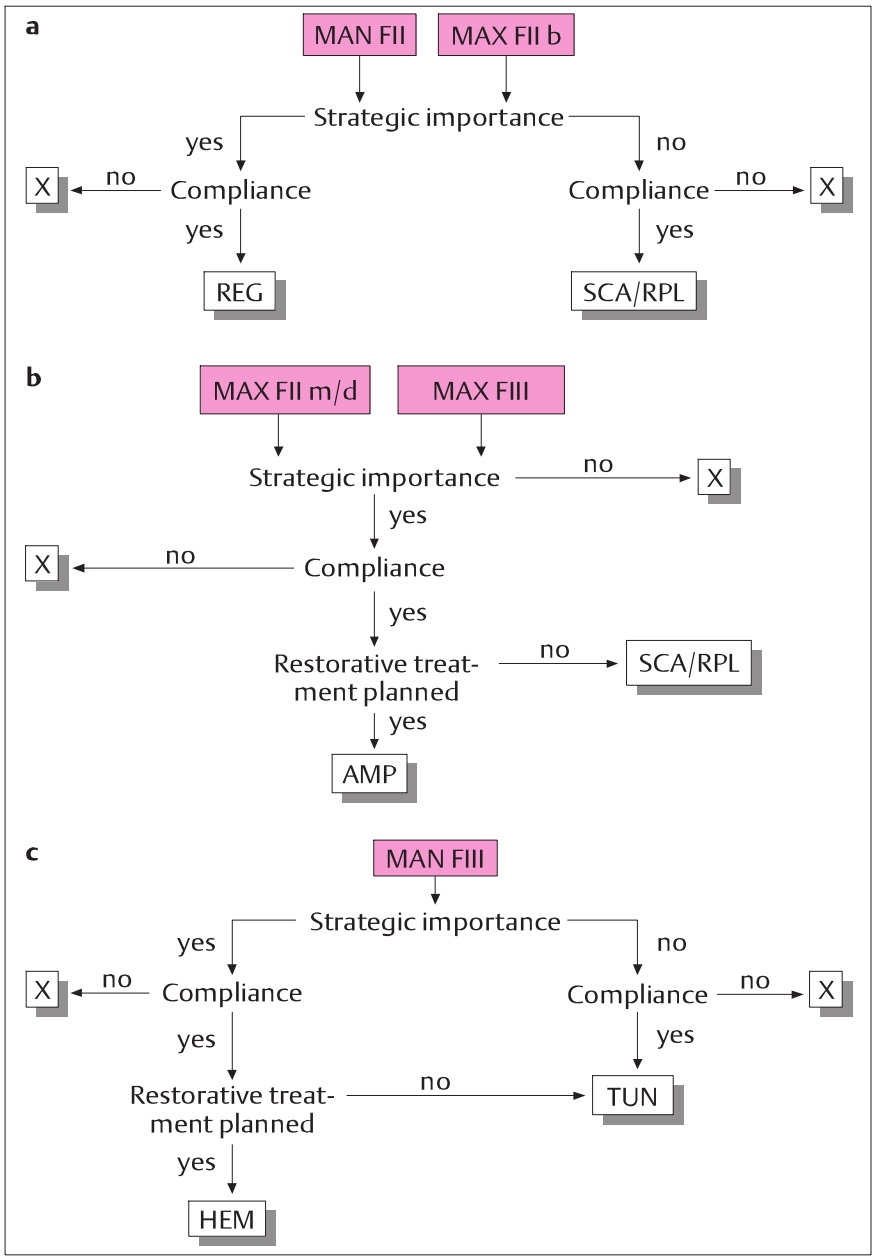


Fig. 11.**35** Decision making in cases of furcation involvement.

a Degree II furcation involvement of mandibular molars (*MAN*) and buccal degree II involvement of maxillary molars (*MAX*). If the tooth is strategically important and compliance is excellent, regenerative treatment should be done. Otherwise, repeated palliative scaling and root planing may improve tooth prognosis. If compliance is poor, the tooth should be extracted in an early treatment phase.

b Mesial and/or distal degree II or through-and-through furcation involvement of maxillary molars. If the morphological and topographical situation is favorable, compliance is excellent, and restorative treatment is planned, root amputation can be considered

c Through-and-through furcation involvement in mandibular molars. If no local factors interfering with the treatment are present, restorative measures are planned, and the patient's compliance is excellent, a hemisectioned tooth can even be incorporated in a fixed partial denture. If the dentition is largely plaque-free, tunnel preparation may be an alternative treatment, especially if restorative treatment can be avoided.

Table 11.**7** Standard instruments of tray for mucogingival surgery

Instruments	Description	Article no., manufacturer
Mouth mirror	Plane, front surface rhodium-coated, ∅ 22 mm	M4C, Hu-Friedy
Periodontal probe	Calibration in 1-mm steps or color coded 3–3–2–3 mm	PCPUNC15 or PCP11, Hu-Friedy
Pliers	• Dressing pliers • Corn pliers • Anatomical tissue pliers • Small curved surgical tissue pliers	• DP18 or DP17, Hu-Friedy • SP20, Hu-Friedy • TP31 or TPG1, Hu-Friedy • TPA, Hu-Friedy
Surgical retractor	Middeldorpf retractor	RSMID2, Hu-Friedy
Scalpel holder	Straight scalpel holder	10-130-05E, Hu-Friedy
Scalers	Sickle scaler H6/H7	SH6/H7, Hu-Friedy
Curettes	• Anterior teeth: Gracey 1/2 • Buccal surfaces of premolars: Gracey 7/8	• SG1/26 or SAS1/2, Hu-Friedy • SG7/86 or SAS7/8, Hu-Friedy
Periosteal elevator		P24GSP or P8D, Hu-Friedy
Scissors	LaGrange gingival scissors	S14, Hu-Friedy
Needle holder	Castroviejo	NH5024R, Hu-Friedy

stable even where there is a narrow band of keratinized tissue.
- Narrow or unattached gingiva is not per se an indication for surgical widening
- However, in some cases this condition may render oral hygiene procedures more difficult.

➤ Indications:
- Decreased vestibular depth in combination with marginal insertion of a lip or cheek frenulum.
- Before root coverage by a coronally repositioned flap.
- Preprosthodontically where there is a subgingival preparation line and thin gingiva.
- Before or during orthodontic treatment to move anterior teeth through the alveolar bone and the attached gingiva is thin or even absent.

➤ Procedure (Fig. 11.**36**):
- Disinfection.
- Local anesthesia.
- Preparation of a periosteal wound bed for a free gingival graft:
  - Supraperiosteal incision with a no. 15 scalpel blade at the mucogingival border,

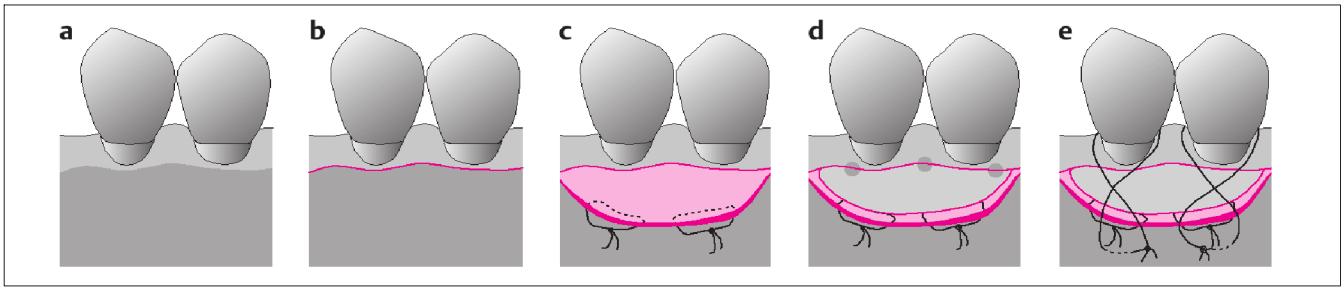


Fig. 11.**36** Free gingival graft.
a Narrow band of gingiva and decreased vestibular depth.
b Supraperiosteal incision at the mucogingival border.
c Supragingival preparation of a recipient site for the graft. The mucosal flap is fixed with two mattress sutures at the periosteum with resorbable suture material.
d The trimmed graft is compressed with a gauze tampon for two minutes and subsequently glued at two or three spots on the coronal margin with tissue glue (Histoacryl).
e Alternatively, the graft can be fixed with two basket sutures

about one tooth-width mesial and distal to the tooth to be treated.
  - Supraperiosteal sharp preparation of a mucoperiosteal flap. *Warning:* beware of injuring the mental nerve in the region of the mandibular premolars.
  - Removal of any remaining muscle fibers with LaGrange gingival scissors.
  - Apical fixation of the mucosa with a horizontal mattress suture (resorbable suture material, 5-0).
  - A template corresponding to the size and shape of the wound bed may be cut out of the sterile suture's wrapping material.
- Local anesthesia at the major palatal foramen.
- Harvesting of a palatal gingival graft from between the second premolar and second molar, or the tuberosity. Harvesting in the region with palatal rugae should be avoided.
  - The template is placed on the palatal mucosa about 2 mm central to the gingival margin.
  - A 1.5-mm-deep incision is made around the template.
  - The template is removed and a 1- to 1.5-mm-thick graft is harvested.
  - *Warning:* Take care not to injure the palatal artery. It is usually located more centrally and runs deeper in the submucosa. If the procedure is carried out correctly, injury can virtually be ruled out.
- The palatal wound is covered with a periodontal dressing (CoePak), which is fixed interdentally.
- The graft is trimmed on the connective tissue side with gingival scissors or a scalpel:
  - to remove any fatty tissue and achieve an even thickness;
  - to adjust the size and shape of the graft to the wound bed.
  - *Note:* In the region of the vestibular fold about 1 mm of the wound should not be covered by the graft.
- The graft should be compressed with a wetted (sterile physiological saline) gauze tampon for two minutes to avoid hematoma formation.
- The graft is then fixed at the gingival margin with two spots of tissue sealant, e.g., Histoacryl
- Alternatively, fixation with a basket suture is possible (Fig. 11.**36e**). *Note:* The graft should not be fixed with interrupted sutures.
- No periodontal dressing should be applied at the grafted site. *Warning:* This might cause postoperative dislocation of the graft.

➤ Usual postoperative infection control and care:
- Mouth rinsing with 0.1–0.2% chlorhexidine solution.
- No toothbrushing in the surgical site for two to three weeks.
- Periodontal dressing at the donor site and sutures are removed after one week; if required, a second dressing is applied.

➤ Wound healing and epithelial differentiation pass through three phases:
- *Initial phase* (days 1–3):
  - Plasma circulation gives graft vitality.
  - Wound transudate enters the graft by capillary action.
  - Epithelium degenerates and is desquamated.
- *Revascularization* (days 2–11):
  - Capillaries proliferate from the wound margins.
  - Formation of anastomoses, build-up of blood circulation.
  - Increasing metabolism within the graft.
  - Ameboid migration of epithelial cells originating from the surrounding tissues onto the vital graft surface.
- *Maturation* (up to the end of the sixth week and thereafter):
  - Reduction of the number of blood vessels within the graft.
  - Epithelial maturation, formation of a keratinized layer.
  - Epithelial differentiation under the influence of the connective tissue beneath.

➤ Critical assessment:
- During harvesting care must be taken to have the graft of an even thickness. *Note:* Grafts that are too thin (less than 0.7 mm) have a strong tendency to shrink.
- Grafts of a defined thickness can be harvested with a handheld or special machine-driven mucotome. Disadvantages:
  - The grafts are rectangular.

- Trimming them to the size and shape of the wound bed involves considerable tissue waste.

– The palatal wound sometimes causes complaints.
– Free gingival grafts have a rather limited range of indications but a high success rate if these are observed:
  - In some cases, a "creeping attachment" (coronal proliferation of the gingiva) may be observed after elimination of non-physiological muscle pulls.
  - *Note:* Due to their orthokeratinization, grafts harvested from the hard palate are usually of a different color to the surrounding tissues.

➤ Supramarginal placement of a free gingival graft for root coverage may lead in some cases to an acceptable result. Procedure:
– Deepithelization of the surrounding tissue
– Harvesting of a fairly thick gingival graft (1.5–2 mm)
– After thorough scaling of the root surface, supramarginal placement of the graft, which must be compressed on the root for several minutes.
– Fixation with a basket suture (cf. Fig. 11.**36e**).

➤ Vestibular extensions without free gingival grafts (e.g., the Edlan–Mejchar surgical technique) are nowadays rarely performed; there might be an indication preprosthodontically to improve the situation for removable dentures.

Gingival Recessions

➤ Multifactorial condition:
– *Primary causes:*
  - Inflammation
  - Trauma to the gingival margin
– *Predisposing factors:*
  - Tooth positioned prominently in the dental arch
  - Dehiscence or fenestration of the alveolar bone
  - Orthodontic tooth movement in a labial direction through the alveolar bone
  - Remodeling of the buccal periodontium following proximal attachment loss

➤ To treat gingival recessions, oral hygiene practices must first be checked. Frequently there is a discrepancy between:
– their intensity and frequency and
– their effectiveness.

➤ Therefore, an optimal oral hygiene must first be established. The patient should be enabled to remove bacterial deposits from the tooth surfaces effectively with appropriate effort and without injuring the gingival tissues.

➤ Following this hygiene phase, the situation should be carefully documented:
– Intraoral photographs are taken.
– Stone model casts are manufactured which document any frenula and their position, the vestibular fold, and gingival recessions.
– A recession chart (cf. Fig. 7.**10**) is filled in.

➤ After this, corrective mucogingival surgery may in appropriate cases be planned.

Lateral Sliding Flap

➤ Indications for a lateral sliding flap (Grupe and Warren 1956):
– localized, shallow Miller class I or II recessions with adequately wide gingiva lateral to the tooth to be treated,
– especially at anterior teeth in the maxilla where there is sufficient vestibular depth.

➤ Contraindications:
– Miller class III or IV recessions
– Several neighboring teeth with recessions
– Generally shallow gingiva
– Decreased vestibular depth, especially in relation to mandibular teeth.

➤ Procedure (Fig. 11.**37**):
– Disinfection.
– Local anesthesia.
– Thorough scaling and root planing:
  - to remove any microbial deposits;
  - to freshen the root surface that has been exposed to the oral environment.
– If considered necessary, the root surface is conditioned:
  - to remove the smear layer;
  - to demineralize the dentin surface and disclose its collagen matrix.
  - E.g., conditioning of the root surface with cotton pledges soaked in citric acid (pH 1) or saturated tetracycline solution. *Note:* In comparative clinical studies, no superiority was observed after root conditioning.

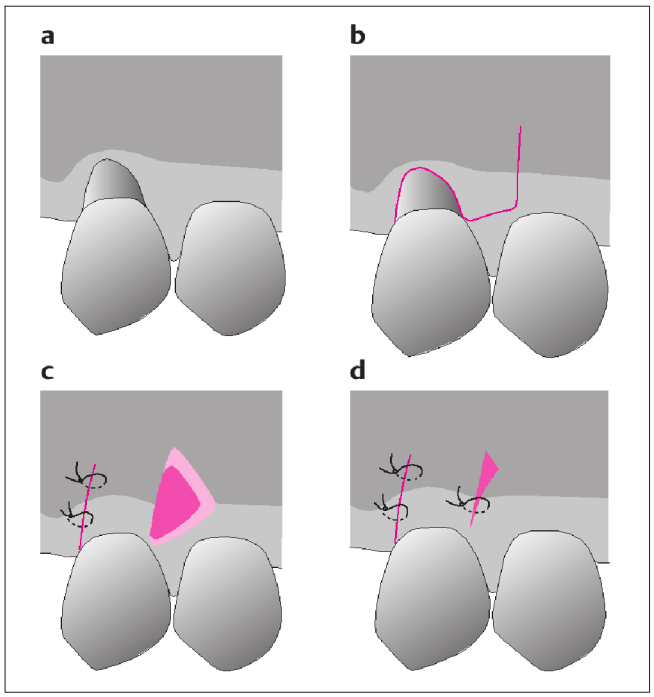


Fig. 11.**37** Lateral sliding flap.
a Localized recession with sufficiently wide gingiva lateral to the neighboring tooth.
b Thorough scaling of the root surface which has been exposed to the oral environment. If necessary, it may be conditioned with citric acid (pH 1) or saturated tetracycline solution. An intracrevicular incision is made at the tooth to be treated, a horizontal incision in the area of the attached gingiva, and a vertical, slightly oblique incision into the vestibulum.
c A mucoperiosteal flap is prepared. The periosteum is dissected at the base of the flap and the flap rotated without tension to cover the recession. The wound margins around the exposed bone are carefully undermined.
d Virtually complete wound closure

- Incisions:
  - Intracrevicular incision in the area of the recession to remove the junctional epithelium.
  - Horizontal incision within the attached gingiva lateral (usually distal) to the tooth to be treated.
  - Vertical, slightly oblique incision into the alveolar mucosa; care should be taken to reveal a wide flap base.
- Preparation of a mucoperiosteal flap:
  - Mobilization beyond the mucogingival border
  - Dissection of the periosteum at the flap base and preparation of a split thickness flap
  - Tension-free coronal/lateral rotation of the flap to cover the recession
- Fixation of the flap with interrupted sutures.
- Careful undermining of the tissues close to the exposed bone surface will in most cases allow wound closure even in the area of the vertical incision.
- No periodontal dressing is placed.

➤ Usual postoperative infection control and care.

Coronally Advanced Flap After Vestibular Extension with Free Gingival Graft

➤ Indications for a coronally advanced flap according to Bernimoulin et al. (1975):
- Localized or several neighboring Miller class II recessions in combination with decreased vestibular depth.
- *Note:* In Miller class I recessions, deepening of the vestibulum is not necessary.

➤ Contraindication:
- Miller class III or IV recessions.

➤ In a first operation, vestibular extension is carried out by free gingival graft (cf. Fig. 11.**36**):
- *Note:* The possibility of creeping attachment after removal of muscular pulls should be taken into account
- Three months later, root coverage is done by a coronally repositioned flap.

➤ Procedure (Fig. 11.**38**):
- Disinfection.
- Local anesthesia.
- Thorough instrumentation and, if considered necessary, chemical conditioning of the root surface.
- The new tip of the papilla is identified as follows:
  - The recession depth is determined with a periodontal probe.
  - This amount is subtracted from the present tip of the papilla at the surgical site.
- Incisions:
  - Intracrevicular incision in the area of the recession to remove the junctional epithelium.

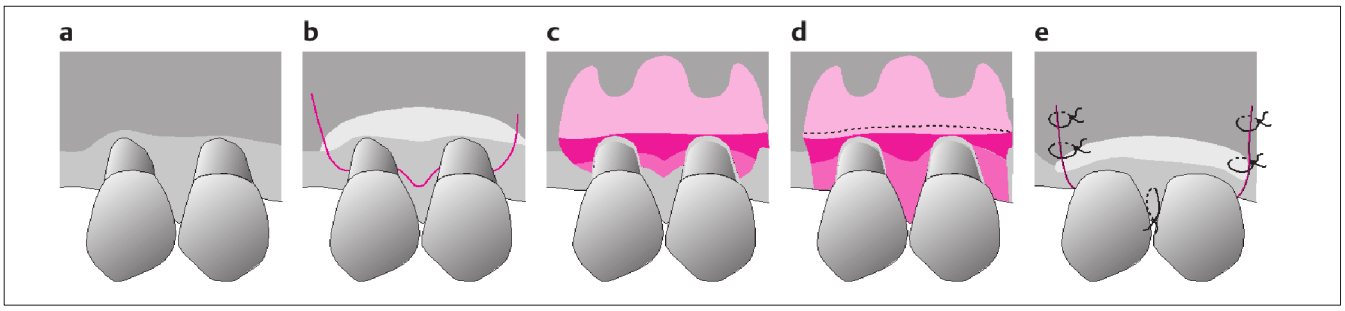


Fig. 11.**38** Coronally advanced flap.
a Two neighboring recessions with narrow gingiva and decreased vestibular depth.
b Situation about three months after vestibular extension with a free gingival graft (cf. Fig. 11.**36**). Incision lines for mobilization of a trapezoidal flap. A new papillary tip is defined according to the depth of the recession. Vertical incisions into the vestibulum bound the flap laterally.
c A mucoperiosteal flap is mobilized and the papillae deepithelialized to create an appropriate wound bed.
d The periosteum is dissected at the base of the flap and the flap advanced without tension for root coverage.
e The wound is closed with interrupted sutures for vertical incisions and a vertical mattress suture interdentally

- Interdentally, horizontal incisions are made circumcising the new papillary tips.
- Two vertical, slightly diverging incisions into the alveolar mucosa bound the surgical site.

– Preparation of a mucoperiosteal flap:
- Mobilization beyond the mucogingival border
- Dissection of the periosteum at the base of the flap and preparation of a split-thickness flap
- Deepithelization of the papillae
- Tension-free, coronal advancement of the flap to cover the recession(s)
- Compression of the flap for about two minutes with a gauze tampon soaked in physiological saline

– Careful wound closure:
- Interrupted sutures for lateral incisions
- Vertical mattress suture (cf. Fig. 11.**11**) interdentally

– No periodontal dressing is placed.

➤ Usual postoperative infection control and care.

Coronally Advanced Flap with Connective Tissue Graft

➤ Indications for a coronally advanced flap according to Langer and Langer (1985):
– Localized deep Miller class I or II recessions
– Especially with thin gingiva

➤ Contraindication:
– Miller class III or IV recessions.

➤ Procedure is similar to that in the coronally advanced flap according to Bernimoulin et al. (1975), but without preceding surgical deepening of the vestibulum.

➤ Sufficiently thick connective tissue grafts can be harvested from three areas of the masticatory mucosa of the hard palate (Fig. 11.**39**):
– Premolar area
– Area of the second molar
– Tuberosity
– *Note:* There is an anatomical barrier in the region of the palatal root of the first molar:
- Rather thin mucosa, not suitable for graft harvesting.
- Separates the part of the hard palate containing minor salivary glands (extends up to the soft palate) from the part containing adipose tissue (in the premolar region).

➤ Recommended procedure for harvesting a palatal connective tissue graft (Müller et al. 1998):
– Disinfection.
– Local anesthesia at the major palatal foramen.
– Incisions:
- Perpendicular to the alveolar bone, a 15-mm-long incision is made about 2 mm distant of the palatal gingival margin.
- Thereafter, a semilunar undermining incision dissects the main part of the lami-

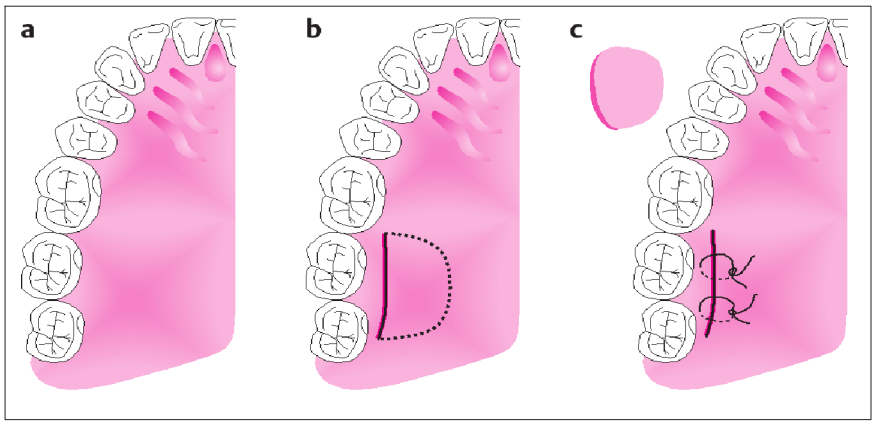


Fig. 11.**39** Harvesting connective tissue grafts.
a Topography of the hard palatal mucosa. Suitably thick mucosa is found in the premolar region and in the region of the second and third molars. A barrier in the region of the palatal root of the first molar usually prevents harvesting of connective tissue of sufficient thickness.
b Harvesting of a connective tissue graft from the more distal area. The first incision is made perpendicular to the tooth axis up to the surface of the alveolar bone. A second, undermining incision is made parallel to the bone surface. After mobilization the circumcised graft is carefully removed.
c Suturing with interrupted sutures

na propria/submucosa from a thin layer of connective tissue covered by epithelium.
  - The graft is mobilized with a small periosteal elevator and removed with tissue pliers.
  - The graft is trimmed.
- Alternatively, two lateral incisions bounding the first incision may be made: the so-called trapdoor flap. *Warning:* Be careful not to injure the major palatal vessels.
- Incisions made with a special double-blade scalpel developed by Harris yield evenly thick connective tissue grafts with a small epithelial collar.

➤ After thorough root instrumentation and, if considered necessary, chemical conditioning, the graft is fixed on the recession with resorbable sling sutures.
➤ The mucoperiosteal flap is mobilized coronally and fixed tension-free with vertical mattress sutures.
➤ The palatal wound is closed with interrupted sutures.
➤ Usual postoperative infection control and care.

Semilunar Coronally Repositioned Flap

➤ Indications for a semilunar coronally repositioned flap according to Tarnow (1986):
- Localized, shallow (not more than 3 mm) Miller class I recessions
- Especially at maxillary teeth

➤ Contraindications:
- Deep recessions of more than 3 mm
- Miller class II, III, or IV recessions

➤ Procedure (Fig. 11.**40**):
- Disinfection
- Local anesthesia
- Thorough instrumentation and, if considered necessary, chemical conditioning of the root surface
- Incisions:
  - Intracrevicular incision in the area of the recession to remove the junctional epithelium
  - Semilunar incision apical to the recession within the alveolar mucosa. The distance between the incision and the soft tissue margin should correspond to the depth of the recession plus a further 2–3 mm
  - Undermining incision to prepare a split-thickness flap
- Tension-free coronal advancement of the split flap
- Compression of the flap for two to three minutes with a gauze tampon soaked in physiological saline
- No suturing
- Periodontal dressing (CoePak or Barricaid) to protect the apical wound

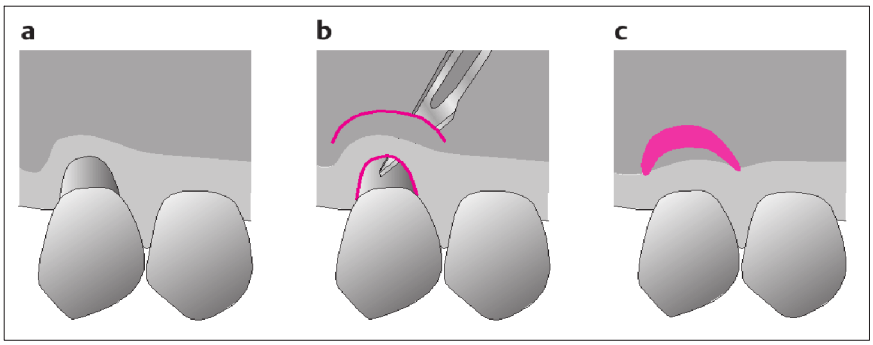


Fig. 11.**40** Semilunar coronally repositioned flap.
a Localized Miller class I recession. The exposed root surface is scaled and, if necessary, conditioned with citric acid (pH 1) or saturated tetracycline solution.
b Semilunar incision in the vestibulum. The distance between the incision and the gingival margin should exceed the recession depth by 2–3 mm. This is followed by sharp preparation of a split-thickness mucosal flap, marginally perforating the gingival sulcus.
c Coronal mobilization and compression of the tissue with a gauze tampon for about three minutes. No suturing of the apical wound

➤ Usual postoperative infection control and care.

Envelope Technique

➤ Indications for the envelope technique according to Raetzke (1985):
- Localized, rather shallow Miller class I or II recessions
- Especially with thin gingiva

➤ Contraindications:
- Deep recessions (more than 3 mm)
- Miller class III or IV recessions

➤ Procedure (Fig. 11.**41**):
- Disinfection
- Local anesthesia
- Thorough instrumentation and, if considered necessary, chemical conditioning of the root surface
- Incisions:
  - Intracrevicular incision to remove junctional epithelium
  - Sharp undermining preparation of a supraperiosteal pouch around the recession site
- Harvesting of a connective tissue graft from the masticatory mucosa of the hard palate
- Placement of the trimmed graft into the prepared pouch (“envelope”)
- If required, fixation with tissue sealant (Histoacryl)
- Palatal wound closure with interrupted sutures

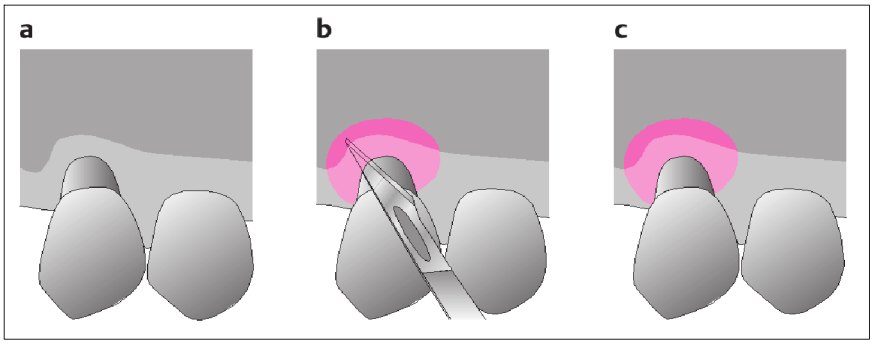


Fig. 11.**41** Envelope technique.
a Localized Miller class I recession. The exposed root surface is scaled and, if necessary, conditioned with citric acid (pH 1) or saturated tetracycline solution.
b Intracrevicular incision to remove the junctional epithelium. Undermining, supraperiosteal preparation of a pouch (“envelope”) for the connective tissue graft.
c The major part of the graft lies within the pouch. Fixation with Histoacryl tissue glue

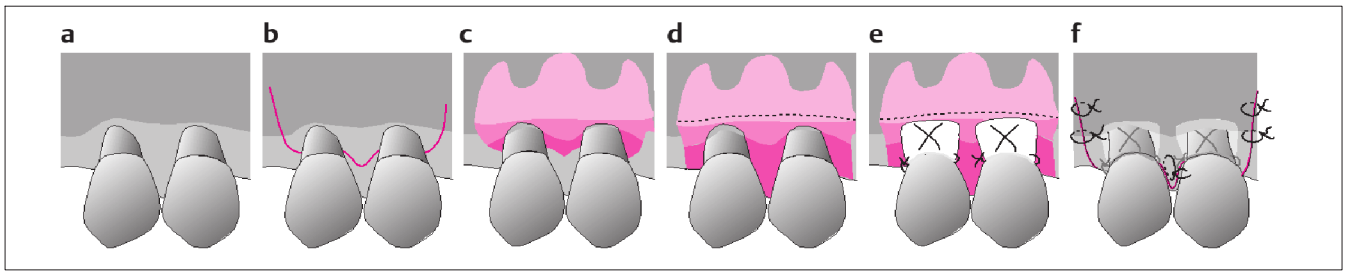


Fig. 11.**42** Root coverage employing guided tissue regeneration.

a Neighboring Miller class I recessions. The exposed root surface is scaled and, if necessary, conditioned with citric acid (pH 1) or saturated tetracycline solution.

b–d Incisions, flap mobilization, and periosteal dissection as for a coronally advanced flap.

e The titanium-reinforced ePTFE membrane is trimmed and fixed with sling sutures.

f The flap is coronally advanced and fixed in a tension-free manner with a vertical mattress suture (interdentally) and interrupted sutures (lateral releasing incisions). The membranes must be surgically removed after four to six weeks

➤ Usual postoperative infection control and care.

Guided Tissue Regeneration

➤ Specially designed membranes are used to provide and maintain sufficient space for the proliferation of cells originating in the surrounding periodontal ligament:
- – Simple ePTFE membrane bent with a sling suture, or ePTFE membrane reinforced with a titanium cross:
  - • To avoid collapse of the membrane on the root surface.
  - • *Disadvantage:* the membrane has to be removed after four to six weeks in a second operation.
- – Special resorbable membranes with space-keeping function: Guidor PPS.

➤ Indications:
- – Localized or neighboring, deep (3 mm or more) Miller class I or II recessions.
- – Especially at maxillary canines and premolars.
- – Possibly: shallow Black class V cavity (the restoration might be exploratively removed).

➤ Contraindications:
- – Shallow recessions
- – Decreased depth of the vestibulum
- – Miller class III or IV recessions

➤ Procedure (Fig. 11.**42**):
- – Disinfection
- – Local anesthesia
- – Thorough instrumentation and, if considered necessary, chemical conditioning of the root surface:
  - • To freshen the root surface which has been exposed to the oral environment
  - • To remove the smear layer with citric acid (pH 1) or saturated tetracycline solution
  - • To create a concave root surface, e.g., with rotating diamond-coated fine (40 µm) and extra-fine (15 µm) burs.
- – Defining new papilla tips:
  - • The recession depth is determined with a periodontal probe.
  - • This amount is subtracted from the present tip of the papilla at the surgical site.
  - • Intracrevicular incision in the area of the recession to remove junctional epithelium.
  - • Interdentally, horizontal incisions are made circumcising the new papilla tips.
  - • Two vertical, slightly diverging incisions into the alveolar mucosa laterally bound the surgical site.
  - • Intracrevicular incision on the palatal/lingual aspect.
- – Preparation of the mucoperiosteal flap:
  - • In the vestibulum, the trapezoidal flap is raised beyond the mucogingival border.
  - • Palatally/lingually, only papillae are raised.
  - • The periosteum is dissected at the base of the flap.
  - • Deepithelization of the papillae.

- A nonresorbable, titanium-reinforced membrane or special resorbable membrane (e.g., Guidor) is selected:
  - The membrane is trimmed until it overlaps the bony margins apically by about 2 mm, laterally by 1 mm.
  - The membrane is fixed with sling sutures (integrated in the Guidor membrane).
- Tension-free coronal advancement of the vestibular flap.
- Careful wound closure:
  - Interrupted sutures for lateral incisions.
  - Interdentally, a vertical mattress suture (cf. Fig. 11.**11**) is made (ePTFE suture material).
- No periodontal dressing is placed.

➤ Postoperative infection control:
- *Note:* Antibiotics are not prescribed on a routine basis
- The patient should rinse his/her mouth twice daily with a 0.1-0.2% chlorhexidine solution for four to six weeks.
- During the first two postoperative weeks, wound healing is checked every two or three days; any plaque present at the gingival margin is carefully removed.
- If the membrane is exposed, the patient is advised to apply a 1% chlorhexidine gel twice daily. Secondary surgical coverage should *never* be performed.

➤ If a resorbable membrane was placed, sutures are removed after two to three weeks.

➤ A nonresorbable membrane has to be removed in a second operation after four to six weeks:
- Intracrevicular incision
- Raising of a small mucoperiosteal flap
- Cutting of the sling suture
- Removal of the membrane with tissue pliers
- Careful curettage of the inner surface of the flap
- Coronal repositioning and fixation of the flap with interrupted sutures

Critical Assessment

➤ Especially during the past 20 years, new methods and modifications for root coverage have been developed:
- Miller class I and II recessions can be covered with different methods. Root coverage may vary between 80% and 100%.
- Where there are Miller class III (interproximal attachment loss) and class IV recessions (interdental bone loss up to a level apical to the marginal tissue recession) satisfactory results cannot be expected.
- Deep and, especially, wide recessions cannot be covered as successfully as shallower and narrower ones.
- On the basis of a recent metaanalysis, guided tissue regeneration may lead to acceptable results of about 75% root coverage, with complete coverage in about 40%.
- On the other hand, especially for shallow recessions (<3 mm), connective tissue grafts may yield better results.

➤ Quality of the new dentogingival connection:
- After guided tissue regeneration, new cementum and a new connective tissue attachment on the previously exposed root surface can be expected.
- With all other methods for root coverage, wound healing results essentially in the formation of a long junctional epithelium.
- However, due to the fairly large area from which regenerative processes can take place (apical and lateral borders of the recession), some new cementum and new connective tissue attachment can also be expected after coronally advanced flaps.

➤ Increasing width and thickness of the gingiva:
- Can usually be observed: after coronally advanced flaps, especially when preceded by widening of the vestibulum with a free gingival graft; after placement of connective tissue grafts.
- May be particularly important in patients with a persistent tendency to injure the gingival tissues
- Widening of the gingiva is not to be expected after guided tissue regeneration; resorbing granulation tissue leads to reversible thickening of the tissue during the resorption period.

Frenectomy

➤ Surgical correction of lip and cheek frenula (Fig. 11.**43a**):
- V-Y plasty
- Z plasty
- Free gingival graft

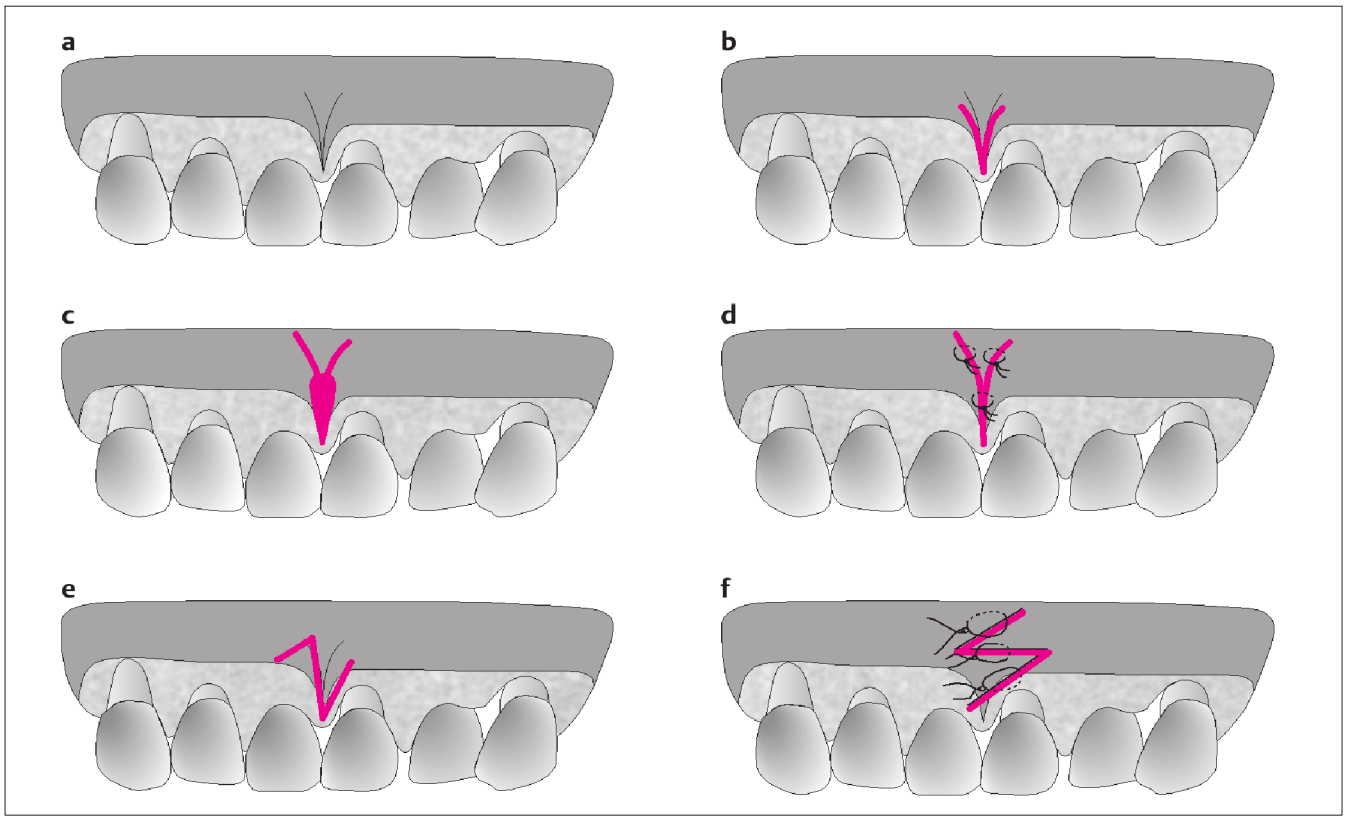


Fig. 11.**43** Frenectomy.
a The marginally inserting, interfering frenum of the lip is planned to be removed.
b–d V-Y plasty. V-shaped circumcision (**b**); supraperiosteal preparation of a mucosal flap and apical repositioning (**c**); apical fixation of the two resulting sides of the Y with interrupted sutures.
e, f Z plasty. Z-shaped supraperiosteal incision. Preparation of two triangular mucosal flaps (**e**); exchange of the flaps and fixation with interrupted sutures (**f**)

- Indications:
  - Before and during orthodontic diastema closure
  - Marginally inserting frenula interfering with
    - effective oral hygiene;
    - wound healing after periodontal surgery.
- Procedure for V-Y plasty (Fig. 11.**43b-d**):
  - Disinfection
  - Local anesthesia
  - Incision:
    - V-shaped circumcision of the frenum of the lip
    - Supraperiosteal preparation of a mucosal flap and dissection of muscle pulls
  - Apical mobilization of the flap and fixation with interrupted sutures
- Procedure for Z plasty (Fig. 11.**43e-f**):
  - Disinfection
  - Local anesthesia
  - Z-shaped incision through the frenum
  - Supraperiosteal preparation of two triangular flaps
  - Exchange of the position of the flaps
  - Fixation with interrupted sutures
- A periodontal dressing is not necessary for either technique.
- Usual postoperative infection control and care.

Occlusal Therapy

General Remarks

- Occlusal forces may affect various structures of the stomatognathic system:
  - Marginal and apical parts of the periodontium

- Pulp–dentin complex
- Occlusal surfaces of the teeth
- Temporomandibular joint
- Neuromuscular system

➤ Causes of occlusal traumatism, which may occur in combination, include:
- *Psychological stress*, frequently resulting in parafunctions such as clenching and bruxism
- *Premature occlusal contacts*
- Other forms of nonphysiological strain, e. g., inappropriate prosthesis.

➤ Experiments in animals and humans have shown the following:
- Occlusal trauma cannot induce inflammatory lesions in the marginal periodontium.
- However, excessive jiggling forces may accelerate the progression of existing periodontitis.
- Increased tooth mobility may have a negative effect on the ecological conditions in the pocket; especially, periodontal pathogens may proliferate.
- *Note:* The pathogenetic significance of occlusal trauma for the periodontium is considered rather low.

➤ Signs of existing periodontal occlusal trauma are:
- Marked deviation of the tooth during articulation.
- Progressively increasing tooth mobility.
- Widening of the periodontal ligament space at the alveolar crest and, sometimes, around the apex.
- *Note:* Increasing tooth mobility due to occlusal interference is essentially regarded as a sign of *physiological compensation*; a tooth with increased mobility can evade excessive forces during articulation.

➤ The deviation of a tooth crown can be measured with Mühlemann's *periodontometer*. Tooth mobility may be distinguished into two sorts:
- *Desmodontal (initial) tooth mobility:* small forces up to 1 N lead to linearly increasing deviation of the crown of between 0.05 and 0.1 mm due to displacement of the tooth within the periodontal ligament space.
- *Periodontal (secondary) tooth mobility:* larger forces up to 5 N lead to distortion and compression of the alveolar process. The crown may be displaced by 0.08–0.15 mm. Secondary tooth mobility:
  - varies among teeth: incisors > canines > premolars > molars;
  - is greater in children than in adults;
  - is greater in women than in men;
  - increases toward the end of pregnancy.

➤ Elimination of occlusal trauma as part of periodontal therapy includes:
- Minor orthodontic measures
- Temporary wearing of an occlusal splint to minimize the influence of potentially harmful parafunctions
- Occlusal adjustment
- Semipermanent or permanent splinting of teeth
- Stabilization of remaining teeth and replacement of lost teeth by definitive restorative treatment

Occlusal Splint

➤ For short-term relief of parafunctional problems in patients with tense jaw, face, and neck muscles:
- Relaxation of the tense muscles.
- Stabilization of the occlusion in centric relation.
- *Note:* Before occlusal surfaces are irreversibly altered by, e. g., selective grinding or extensive restorations, occlusal therapy with appropriate splints should be carried out.

➤ Manufacturing of a resin acrylic splint (e. g., Michigan splint) for the maxilla (in cases of Angle class III malocclusion, mandibular splints are also possible):
- Axis–orbital mounting of stone model casts in a semiadjustable articulator.
- Care must be given to produce only a small increase in the vertical occlusal dimension, which must be acceptable to the patient.
- Centric stops and canine guidance are waxed up.
- Interference-free gliding of the teeth with freedom in centric must be achieved.
- The splint is processed to acrylic resin, trimmed, and polished.

➤ The splint should be worn during the first three weeks day and night.

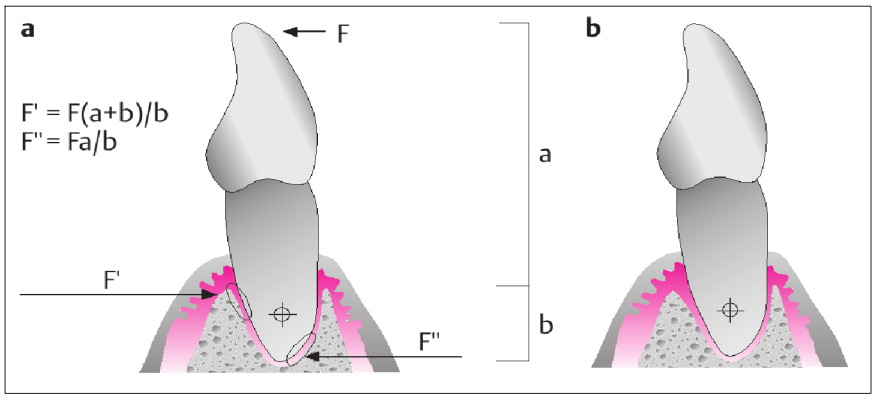


Fig. 11.**44** Secondary occlusal trauma.
a If the relation between the length of the clinical crown (*a*) and the total length of the tooth (*a*+*b*) is unfavorable, considerable forces may arise at the alveolar crest and at the apex during normal function
b If periodontal infection is under control, compensatory bone resorption in the region of the alveolar crest and the tooth's apex mean that in this situation, too, the tooth can evade the detrimental forces and attachment loss is not to be expected. Clinically, tooth mobility may be progressive, which may make splinting necessary

Occlusal Adjustment

- Clinical occlusal analysis may sometimes reveal evidence of occlusal trauma:
  - The main symptom is progressive tooth mobility.
  - Further hints are: palpable and visible deviation of the tooth in maximum intercuspation and during articulation.
- Tooth mobility is a function of:
  - the height of the alveolar bone;
  - the width of the periodontal ligament space;
  - the morphology of the root complex.
- In any case, careful differential diagnosis is necessary. *Note:* Teeth which are hypermobile because of advanced bone loss cannot become firm after occlusal adjustment.
- Teeth frequently change their position during the course of progressive periodontitis:
  - Unilateral pressure exerted by granulation tissue in a bony pocket may cause the tooth to move away from the periodontal lesion.
  - The resulting occlusal interference may lead to periodontal trauma, since the tooth is alternately pushed during function into one or the other direction (jiggling).
  - Such interfering contacts must be removed very early during therapy.
- Selective grinding should be achieved only *after* periodontal treatment is finished:
  - Elimination of interferences in centric occlusion
  - Elimination of balancing interferences
  - Harmonizing of working side contacts
- Possible indications for occlusal adjustments include:
  - Prevention of and diminishing excessive parafunctional habits such as clenching and bruxism
  - Harmonizing of the occlusal plane
  - Corrections after orthodontic and before restorative treatment
  - Only rarely: a periodontal indication
- *Note:* An "ideal" occlusion created by selective grinding or restorative procedures is *not* a desirable treatment aim per se.

Semipermanent Splinting

- Increased tooth mobility as a result of a reduced periodontium is acceptable as long as the occlusion is stable and chewing comfort is not impaired.
- An unfavorable relation between the clinical crown and the total tooth length may result in considerable forces at the alveolar crest and at the apex during function (Fig. 11.**44**): so-called *secondary occlusal trauma.*
- In these cases splinting of teeth results in reduction of the periodontal ligament space,

since the detrimental forces are distributed among several teeth.

- Indications for semipermanent splinting:
  - Significant reduction of the tooth-supporting apparatus
  - Progressive tooth mobility
  - Risk of tooth loss during function or treatment
- Procedure:
  - Splinting with composite after etching quite large enamel areas of the tooth to be splinted and neighboring teeth. The splint may be reinforced with carbon fibers, or armed with metal or acrylic mesh.
  - Creation of small, mobile units is of advantage (Fig. 11.**45**); if more than three teeth are united, the risk of fracture (at the firm tooth) increases considerably.
  - Occlusion must be checked carefully; especially for anterior teeth in the maxilla an esthetically unacceptable situation may arise.
  - Periodontal hygiene must not be hampered; cleaning of embrasures with interdental brushes or dental floss (Superfloss) at home must be possible.
- Splints that are not functionally or cosmetically disturbing may remain in situ permanently.

Fig. 11.**45** Splinting of a central and lateral incisor and with composite: a small, still slightly mobile unit is less susceptible to fracture, since teeth are allowed to evade occlusal forces during functional strain. Homecare hygienic measures are possible.

Perioprosthetic Aspects

- Generalized aggressive and advanced chronic forms of periodontitis usually lead to early loss of molars. The remaining teeth frequently exhibit markedly increased tooth mobility.
- In addition to the necessary replacement of lost teeth in order to restore chewing function and to improve esthetics, stabilization of the remaining teeth is a major treatment goal of restorative therapy:
  - *Note:* Increased tooth mobility and advanced loss of the tooth-supporting apparatus are not per se contraindications for fixed restorations.
  - In cases of increased tooth mobility, chewing comfort may be improved considerably by permanently splinting teeth in a fixed partial denture.
  - In patients with a reduced number of teeth and increased tooth mobility, fixed restorations have several advantages compared to removable dentures:
    - Greater stability of the construction.
    - Better distribution of chewing forces.
  - In particular in patients in their 50s and 60s, the possibility of a shortened tooth arch (premolar occlusion) should be seriously considered (Fig. 11.**46**).
  - Occlusal stabilization in patients with unilateral or bilateral shortening of the dental arch may also be achieved by distal cantilevers:
    - If possible as a bilateral fixed partial denture.
    - A unilateral fixed partial denture needs at least two abutments.
    - Abutments with low retention are contraindications.
  - To avoid loss of retention, abutments should be prepared with maximum height and minimum taper:
    - The principle of self-retention must be observed.
    - The inclination of the crowns should be opposite to the dislocating force that acts on the cantilever.
  - In certain cases orthodontic distalizing of a premolar and subsequent insertion of a fixed partial denture may be possible.

Fig. 11.**46** Patient with advanced loss of alveolar bone, especially in the maxilla. Periodontal and restorative treatment is finished

- Fixed restorations should not be combined with oral implants.

➤ Restorative treatment may be conducted about four to six months after completion of periodontal therapy.

➤ Procedure for the manufacturing of fixed bridges in dentitions with marked tooth mobility:
- A two-stage procedure is recommended, by which one jaw is restored first (in this case, e.g., the upper jaw).
- Study model casts are mounted in a semi-adjustable articulator in maximum intercuspation.
- If required, incisor-canine guidance is recorded.
- An acrylic registration plate is made in the articulator.
- Preparation of abutment teeth; the preparation height should amount to more than 4 mm to achieve maximum retention of the crowns:
  - This is usually not a problem in the case of long clinical crowns.
  - However, if there is marked divergence of abutment teeth, devitalization, root canal treatment, and incorporation of post and core may be indispensable
  - The possibility of attachments should be considered in this case.
- Impression making:
  - Double-mix impression material (silicon) may be used in a perforated tray.
  - Hydrocolloid impression material should be avoided: teeth with marked root exposure are at a high risk of irreversible thermal pulp damage.
  - *Warning:* Very firm, addition-type silicon or polyether impression material should not be used; considerable problems may arise during removal of the impression from the oral cavity as well as from the model cast.
- An alginate impression is made of the opposite jaw with a rimlock tray.
- The master model and the opposite jaw model are manufactured.

- *Note:* In the case of very mobile teeth, registration of the cusp tips of the dental arch should not be done in situ but must be performed on the registration plate using the master model:
  - A registration paste (zinc oxide eugenol paste, e.g., Temp-Bond) is applied on the registration plate.
  - The watered master model is placed on the registration plate; an impression of cusps is made with the soft paste.
  - The paste can be allowed to set, e.g., in a water bath.
- After setting of the registration paste, impressions that are too deep are trimmed with a scalpel blade; the fit of the impressions in the patient's mouth is checked.
- In order to minimize an increased vertical dimension, the registration plate is ground from the mandibular side until a small perforation in centric occlusion is noted.
- Centric relation record:
  - On the mandibular side of the registration plate a small amount of heated thermoplastic composite material is applied in the region of the central incisors.
  - The mandible is guided in centric relation towards the inserted registration plate. The frontal stop within the thermoplastic material should just inhibit contact of posterior teeth with the plate.
  - It should be repeatedly checked that the patient easily finds the frontal stop in centric occlusion.
  - Zinc oxide eugenol paste is applied to the mandibular side of the registration plate in the region of the posterior teeth. The maxillary side of the plate is inserted into the patient's mouth, and the patient closes in centric occlusion.
  - Pressure-free moulds of the posterior teeth of the mandible are made in the soft registration paste.
- After the registration paste has set, the plate is removed from the mouth.
  - The frontal stop is discarded with a scalpel blade.
  - Impressions are shortened to a level where only the tips of the cusps remain visible.
- Fit of master and opposite jaw model with the registration plate is checked.
- Model casts are mounted in the articulator:
  - The upper jaw model is mounted using a face-bow.
  - The lower jaw model is mounted using the centric relation record.
  - Mounting is checked with the patient's clinical situation.

➤ In the manufacturing dental laboratory, the increased risk of fracture of the fixed partial denture must be taken into account:

- Bridgework should be of proper dimensions and necessary rigidity, though they must also allow adequate interdental hygiene
- Fixed partial dentures should never be soldered; instead, sliding attachments should be included in the design (functioning as stress breakers).
- Although possible on principle, cantilever bridges should be avoided whenever possible because of an increased risk of failure due to:
  - loss of crown retention;
  - fracture of metal frame;
  - tooth fracture.
- Combinations with implants are questionable.

➤ *Note:* Subgingival crown margins are incompatible with gingival health.

- In areas not exposed to view, supragingival crown margins should be placed, if necessary, after crown lengthening.
- In visible areas, some compromise must be sought for esthetic reasons:
  - Slightly subgingival (about 0.5 mm), technically perfect (marginal imperfections of not more than 50 µm) crown margins are tolerable.
  - The *biological width* must be taken into account: there should be a distance of 2.5–3 mm between the alveolar crest and the margin of the restoration. *Note:* The biological width is smaller in individuals with narrow and thin gingiva.
  - If the biological width is disregarded, attachment loss or recurrent, frequently proliferating inflammation of the gingiva is to be expected.

➤ Failure after incorporation of fixed partial dentures for stabilization of periodontally impaired dentitions is frequently due to technical and biophysical factors:

- Loss of retention of the crown.
- Fracture of the metallic frame.
- Root fracture after incorporation of post and core.
- *Note:* As a general principle, the most important prerequisite for long-term success is control of the periodontal infection.

12 Phase III: Supportive Periodontal Therapy

Risk Assessment, Risk Communication, Risk Management

General Remarks

➤ In patients susceptible to periodontal disease, there is a high risk of renewed infection with *periodontal pathogens*. After completion of the corrective phase of therapy, life-long supportive care should be organized on an individual basis:
- Following periodontal surgery, the progress of healing is followed up weekly or every second week.
- Periodontal reevaluation is carried out after two to three months. Thereafter, *supportive periodontal care* commences, i. e., phase III of periodontal therapy.

➤ Risk assessment, risk communication to the patient, and risk management are the cornerstones of supportive care. The goals of this third treatment phase, which in the past was termed "maintenance therapy," are:
- Risk-related medical/dental history taking and clinical examination.
- Remotivation of the patient, continuous medical support.
- Timely and appropriate intervention in any case of recurrent periodontitis.
- Avoidance of any under- or overtreatment.

➤ *Note:* The long-term success of any periodontal therapy depends less on particular surgical measures or devices than on the quality of supportive periodontal care.

Risk Assessment

➤ Reduction of the deleterious influences of established risk factors is now regarded as central to the therapy of any complex, multifactorial, chronic disease. This is particularly true for the treatment of periodontal disease:
- The onset and progression of periodontitis are largely determined by risk factors:
  - Risk factors are either part of the *causal chain* or *expose* the individual to disease
  - *Note:* Once disease has broken out, elimination of the risk factor does not necessarily lead to healing.
- Risk assessment for recurring periodontitis is usually performed on three levels:
  - *Local risk* assessment
  - *Dentition-related risk* assessment
  - *Systemic risk* assessment

Local Risk Factors

➤ *Redness* and *swelling* of the gingiva are cardinal symptoms of inflammation and should be carefully assessed in any periodontal examination:
- They are visible signs of inflammatory reactions, especially to the supragingival portion of dentogingival plaque;
- For this reason they are not suitable as a diagnostic test for periodontal activity.

➤ *Bleeding on probing* to the bottom of the sulcus or pocket (Fig. 12.**1**):
- Can easily be checked by local examination.
- Bleeding on probing—especially if a pressure-controlled probe is used (about 0.25 N, e. g., ClickProbe)—gives a fairly reliable impression of local inflammation within the sulcus or pocket.
- Bleeding on probing has been used as *diagnostic test* for attachment loss during supportive periodontal care. *Frequent bleeding on probing:*
  - Has a low sensitivity (many false-negative findings) but quite a high specificity (few false-positive findings) in detecting active periodontal disease.
  - Constant bleeding on probing increases the risk of attachment loss about two- to three-fold; absence of bleeding on probing signifies a stable condition.

➤ *Purulent exudate:*
- In the past pus gave its name to the disease of which it was a symptom, pyorrhea alve-

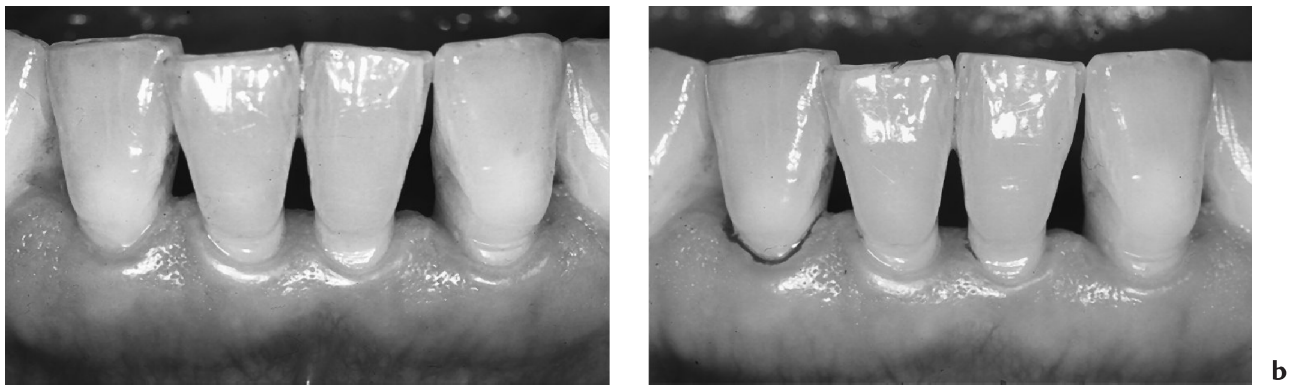


Fig. 12.1 Bleeding on probing.
a Clinically, periodontal situation shows no signs of inflammation.
b Profuse bleeding after probing at the distal aspect of the right lateral incisor. Small bleeding spots are seen distal to the right and mesial to the left central incisor. Frequent bleeding after probing is a rather insensitive but relatively specific test for periodontal activity after therapy

olaris; it is rarely observed during supportive periodontal care.
  - It is an exudate containing predominately polymorphonuclear granulocytes.
  - Because it lacks sensitivity (many false-negative findings), purulent exudate is not a prognostic indicator.
  - If present, it should always prompt therapeutic measures.

➤ *Increased probing depth:* Periodontal probing depths of more than 3 mm are frequently noted after periodontal therapy. Differential diagnosis:
  - Increased probing depth due to a long junctional epithelium after periodontal surgery: no inflammation; especially, no bleeding on probing.
  - Increased probing depth because of presence of a periodontal pocket: inflammatory reaction; especially, bleeding on probing.
  - There is a strongly increased risk of further attachment loss only in residual periodontal pockets that bleed after probing
  - For this reason, the *combined findings* should be recorded: bleeding on probing can be documented in the periodontal chart by underlining (in red) the periodontal probing depth (cf. Fig. 7.**6**).

➤ Local presence of *periodontal pathogens:*
  - Cell numbers $>10^4$ for *A. actinomycetemcomitans* and $>10^5$ for *P. gingivalis* can drastically increase the risk of local attachment loss.
  - However, microbiological tests on individual tooth surfaces are very expensive.

➤ *Furcation involvement* impairs the prognosis of the affected tooth dramatically, especially if accompanied by increased tooth mobility.

➤ Persisting *bony pockets* may have a slightly increased risk (about 30%) of further attachment loss.

Dentition-Related Risk Factors

➤ Proportion of *tooth surfaces covered by supragingival plaque* (after disclosing); proportion of *gingival units that bleed after probing*:
  - During supportive periodontal care excellent oral hygiene is required of the patient.
  - *Note:* In plaque-infected dentitions, a halt in the progression of periodontal disease is not to be expected.
  - The proportion of gingival units that bleed after probing corresponds to the overall inflammatory condition of the tissues.
  - The *relation between the two parameters* (e. g., the plaque–bleeding ratio, or the site-specific association) may better describe the risk of further attachment loss:
    - A low proportion of tooth surfaces covered by plaque but a high proportion of bleeding on probing: high risk.
    - A high plaque proportion and a low proportion of bleeding on probing: quite low risk
    - *Note:* Smokers usually have comparable amounts of plaque and slightly more cal-

culus than non-smokers; bleeding tendency after probing is, on the other hand, reduced: this masking effect of smoking may lead to risk miscalculation.

- Number of *residual pockets*, number of *open furcations:*
  - Both of these expand the habitat for obligately anaerobic periodontal pathogens
  - *Note:* Numerous residual periodontal pockets of 5 mm or more and open, inappropriately treated furcations raise the periodontitis risk for the whole dentition.
- Intraoral load of *periodontal pathogens:*
  - A pooled sample of the four deepest pockets can give an impression of the overall oral load of selected periodontal pathogens such as *A. actinomycetemcomitans*, *P. gingivalis*, *P. intermedia*, *T. forsythia*, or *T. denticola.*
  - Valuable information may be gained, particularly in
    - Aggressive forms of periodontitis.
    - Cases refractory to periodontal therapy.
    - Proof of persistent periodontal infection with *A. actinomycetemcomitans* may have prognostic value, particularly in cases of aggressive and advanced chronic periodontitis.
  - *Note:* Microbiological tests should not be performed on a routine basis. Any possible gain in information must always be set against the considerable costs involved.
- *Tooth loss* due to periodontitis. The risk of further tooth loss is higher if numerous teeth have already been lost due to periodontal disease:
  - *Note:* The decision for or against an extraction is not always based entirely on the prognosis of the particular tooth.
  - This is why the reasons for previous extractions need to be carefully investigated.
- *Bone loss* in relation to age:
  - Advanced, generalized bone loss at a young age signifies a high risk of further attachment loss.
  - On the other hand, mild or moderate bone loss in middle or old age could indicate a low risk.
- Complex restorative treatment contains numerous risks which have to be weighed up regularly:
  - Occlusal instability, especially increasing tooth mobility
  - High risk of fracture of the construction or loss of retention
  - Oral hygiene requirements
  - Tooth fracture
  - Endodontic problems

General Risk Factors

- During supportive periodontal care, aggressive and early-onset forms of periodontitis have to be judged in a different way to more chronic forms of the disease:
  - There might be a genetic predisposition (cf. Table 7.**4**).
  - There might be a persistent infection with periodontal pathogens that are difficult to eliminate, such as *A. actinomycetemcomitans.*
- Some systemic diseases are important risk factors for periodontitis:
  - Therefore, periodical updating of the medical history is mandatory especially in older patients.
  - In the context of supportive periodontal care, cooperation with medical specialists may be necessary:
    - Diagnosis and metabolic control of diabetes mellitus.
    - Close cooperation with the physicians of patients infected with HIV.
    - Hormone replacement therapy for the prevention of menopausal osteoporosis has recently been criticized on the basis of increased other health risks.
- Reduction of smoking, the most important risk factor:
  - Assessment and documentation of smoking status (cf. Table 8.**4**).
  - Medical counseling and assistance, if the patient can be persuaded to quit smoking.

Communication

- Established and presumed risks for the onset and progression of periodontal disease (Table 12.**1**) can be graphically displayed in multidimensional risk diagrams (Fig. 12.**2**):
  - Visualization of individual risks.
  - Improves communication with the patient:
    - Motivation for renewed improvement of oral hygiene.

Table 2.1 Known and presumed risks for development and progression of periodontal disease

Risk	Examples	Relative risks
Etiological factors	• Poor oral hygiene • Periodontal pathogens – *A. actinomycetemcomitans* – *P. gingivalis* – *T. forsythia*	2 2–20
Genetic susceptibility	• IL-1 haplotype • Fcγ-RIIa 131-R/H polymorphism • Fcγ-RIIIb NA1-NA2 polymorphism	2.5–19
Drugs	• Immune suppressive agents – Cyclosporine – Tacrolimus • Ca^{2+} antagonists	
Behavior	Coping with emotional stress	1.5–2
Background variables	• Age • Gender • Race	Increases with age Males > females Blacks > Asians > Caucasians
Systemic diseases	• Diabetes mellitus • Osteopenia/osteoporosis	2.8–3.4, may be higher in noncontrolled populations
External exposure	Smoking	2.5–6, may be higher in young populations
Socioeconomic factors	• Education • Poverty	

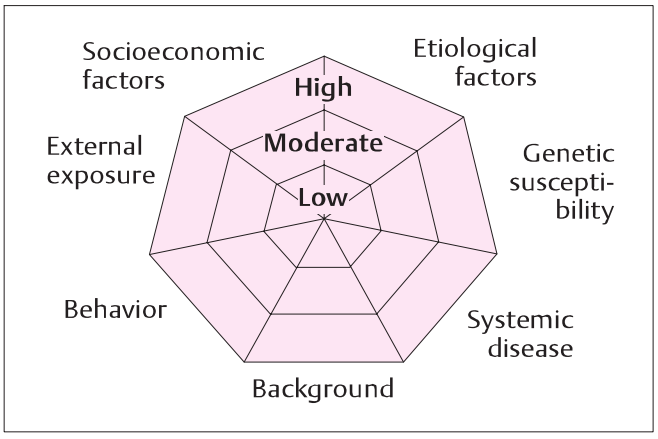


Fig. 12.2 Multidimensional diagram for risk assessment of complex diseases. The different sectors represent clusters of different factors (cf. Table 12.1). Polygonals connect comparably low, moderate, and high risks. This allows the risk in a given case to be more easily visualized. (Adapted from Tonetti 1998).

- (In smokers) alteration of smoking behavior.

– Allows *joint planning* with the patient of how to proceed.
– *Note:* Dentition-related and (superordinated) general risks should be presented in separate risk diagrams.

➤ The long-term compliance of the patient largely depends on his or her understanding the rationale behind the proposed measures.

Risk Management

➤ Supportive periodontal therapy may be carried out within a one-hour appointment:
– Examination (about 15 minutes):
- Update of medical/dental history
- Intraoral, especially periodontal examination (cf. Figs. 7.6 and 7.11)
- Risk assessment

– Detailed counseling (about 15 minutes):

- If required, motivation to improve oral hygiene
- If required, medical support in giving up smoking
- Discussion of further procedure

– Active measures (about 30 minutes):
- Supragingival scaling
- Subgingival scaling only at sites with probing depths of >3 mm and bleeding on probing. *Note:* Subgingival scaling in shallow pockets leads to attachment loss; frequent removal of root substance may result in hypersensitive teeth.
- Open furcations in particular must be carefully débrided with hand instruments and/or ultrasonic or sonic scalers.
- Polishing with toothpaste; local fluoridation with high-dose fluoride gel.

➤ If any surgical procedures are necessary, e.g. if there are more than four bleeding sites with a periodontal probing depth of more than 4 mm, they should be performed separately in a later session.

➤ Recall intervals depend on individual risk:
– For *high risk* patients the interval should be two to three months:
- Combined systemic and behavioral risks.
- Complex and prolonged perioprosthetic treatment.
- Repeated differentiation from recurrent periodontitis.
- *Note:* The proportion of these patients should not exceed 10% in a specialized periodontal practice.

– For *moderate risk* patients, recall intervals of about four to six months are appropriate:
- Certain, easy-to-control risks
- Proportion of these patients in a specialized periodontal practice: about 60%.

– For *low risk* patients yearly sessions are sufficient. *Note:* Overtreatment and leaving patients in uncertainty about disease progress must be avoided.

13 Drug Therapy

Antibiotic and Antimycotic Therapy

Systemically Administered Antibiotics

- Antimicrobial therapy may be justified because of the infectious character of most periodontal diseases, which are caused by a limited number of bacteria.
- A distinction is made between:
  - Specific, i.e., *microbiologically oriented*, chemotherapy following proof of the presence of particular pathogens, and
  - *Empirically oriented* chemotherapy.
- Periodontal infections are not life-threatening diseases and can usually be controlled without the adjunctive use of antibiotics:
  - *Note:* Chronic periodontitis should not be treated by empirical chemotherapy.
  - In cases of aggressive or refractory periodontitis, a microbiological diagnosis may allow specifically targeted antibiotic therapy. Responsible use of antibiotics takes account of:
    - The possible development of *resistance*
    - Antibiotic *toxicity*
    - The risk of *sensitization*
- Before the administration of adjunctive systemic antibiotics, the dentogingival biofilm must be destroyed by thorough scaling and root planing:
  - Structured plaque within the pocket may be as thick as 0.4 mm. It is unlikely that an antibiotic will penetrate these bacterial masses colonizing the root surface.
  - Because of the extremely high number of bacteria in the pocket, the antibiotic appearing in gingival exudate is rapidly used up.
  - Bacterial metabolism and proliferation rate are much reduced in biofilms. Minimum inhibitory concentrations of antibiotics for bacteria residing in biofilms may therefore be 100 to 1000 times higher than concentrations observed in planktonic cultures.
  - Thus, the risk of development of resistance is high.
- Goals of adjunctive systemic antibiotic therapy:

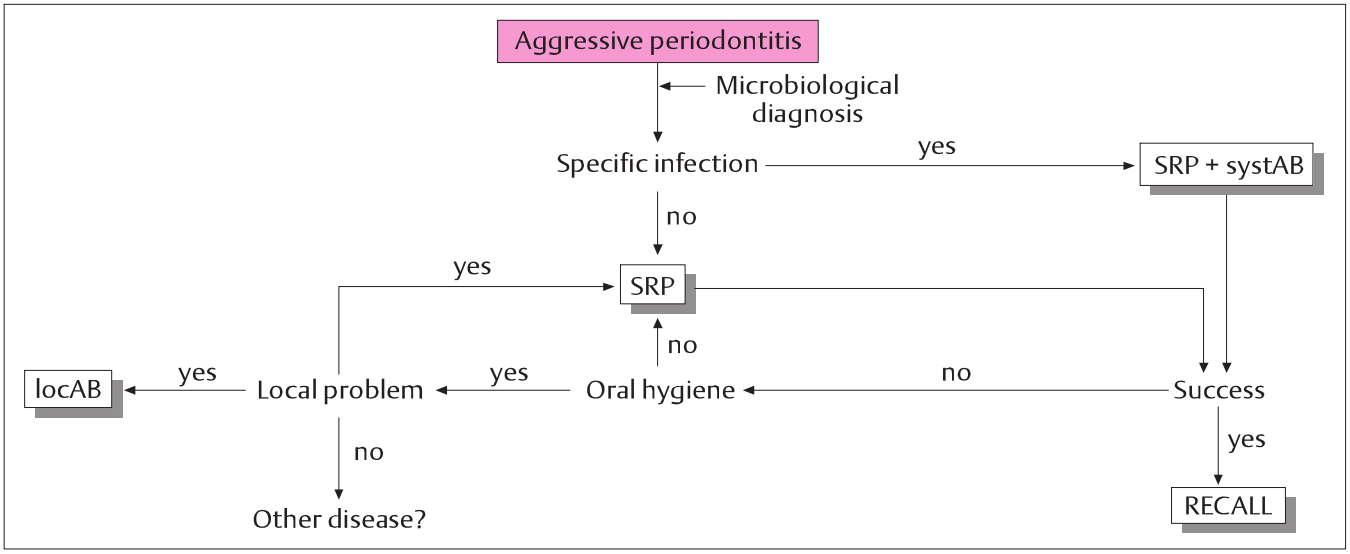


Fig. 13.1 Flow chart for decision making regarding adjunctive systemic (*systAB*) or local antibiotic treatment (*locAB*) in cases of aggressive periodontitis. Care must be taken over the differential diagnosis versus other diseases (e.g., malignancy, Langerhans cell histiocytosis, mucocutaneous disorders, inflammatory alterations associated with corrosion phenomena)

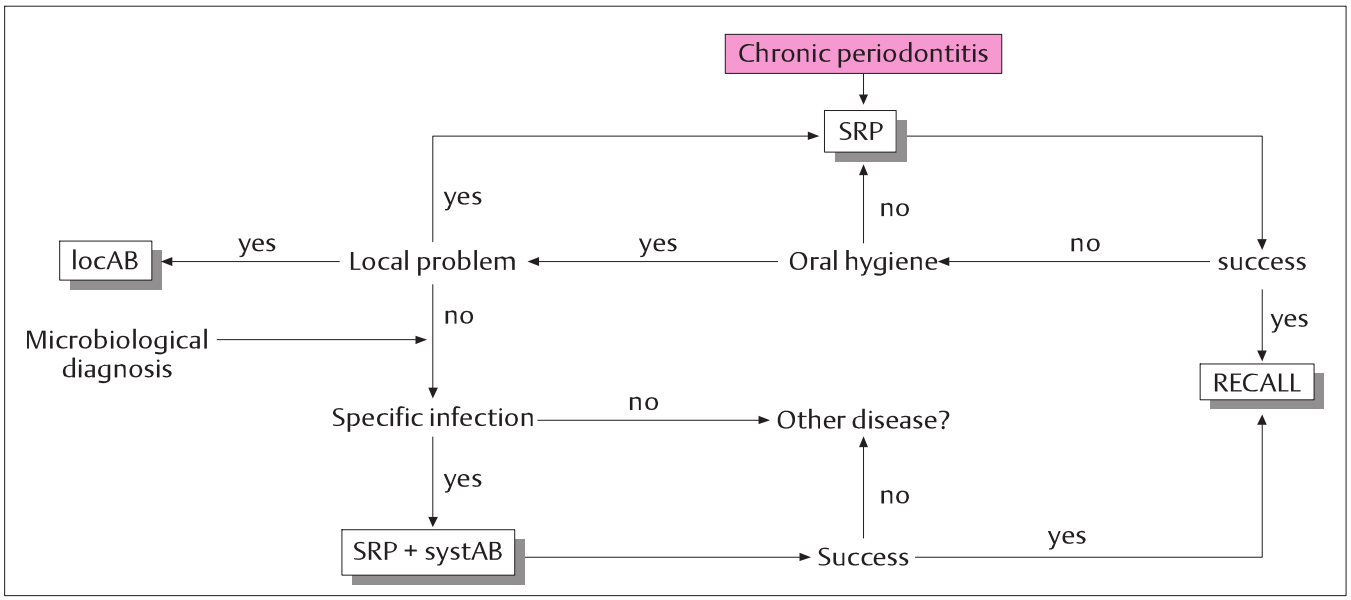


Fig. 13.**2** Flow chart for decision making regarding adjunctive antibiotic treatment in cases of chronic periodontitis

- Reduction or elimination of specific periodontal pathogens.
- If possible, the physiological flora should not be affected.
- Superinfections should be avoided.

➤ Indications:
- Aggressive (Fig. 13.**1**), especially early-onset, periodontitis.
- Refractory chronic periodontitis (Fig. 13.**2**).
- Necrotizing ulcerative gingivitis/periodontitis, especially with fever and/or lymphadenitis.
- Periodontal abscess, especially with fever and/or lymphadenitis.
- Severe forms of generalized periodontitis in combination with systemic diseases:
  - Dysfunction of polymorphonuclear granulocytes
  - Diabetes mellitus
  - HIV infection and <200/mm^3 CD4 cells
- For indications for antibiotic prophylaxis, see Chapter 8.

➤ In cases of chronic periodontitis, the first step is to attempt to control the infection by conventional therapy. If this fails, a careful analysis should be carried out and the answers to the following questions be critically assessed:
- Is the oral hygiene of the patient sufficient?
- Have teeth with furcation involvement been treated properly?
- Is the patient a heavy smoker?
- Is there any underlying systemic disease?

➤ An antibiotic which is adjunctively employed in periodontal therapy should have the following properties:
- *Specificity:* antibiotics that are of great importance in general medicine should not be used to fight periodontal infections.
- *Efficacy:* microbial diagnosis (e.g., culture) should be combined with susceptibility testing. Antibiotics should be bactericidal rather than merely bacteriostatic.
- *Substantivity:* the appropriate antibiotic should appear at sufficient concentration at the site of action (i.e., the periodontal pocket).
- *Safety:* low toxicity and low risk of sensitization.
- *Oral administration:* parenteral and intramuscular administration is not usually tolerated in dental practice.
- *Stability.*

➤ The following antibiotics may be used as an adjunct to conventional periodontal therapy (Table 13.**1**). Relevant contraindications must always be observed:
- *Penicillins:*
  - Interfere with bacterial cell wall synthesis.
  - Exhibit in vitro antibacterial activity against most periodontal pathogens.

Table 13.1 Recommended dosages for adjunctive systemic antibiotic therapy

Antibiotic	Dosage for adults (70 kg)	Duration of medication
Tetracyclines		
• Tetracycline HCl	4 × 250 mg/day	14–21 days
• Doxycycline HCl	1 × 200 mg/day, afterwards 1 × 100 mg/day	1 day, 13–20 days
• Minocycline HCl	1 × 200 mg/day	14–21 days
Metronidazole	3 × 400 mg/day	7–10 days
Amoxicillin/clavulanic acid (Augmentin)	3 × 500 mg/day	7–10 days
Ciprofloxacin	2 × 500 mg/day	7–10 days
Clindamycin	4 × 300 mg/day	7 days
Azithromycin	2 × 250 mg/day	3 days
Combinations		
• Metronidazole/ amoxicillin	3 × 400 mg/day 3 × 500 mg/day	7–10 days
• Metronidazole/ ciprofloxacin	2 × 500 mg/day 2 × 500 mg/day	7–10 days

- However, pocket exudate contains generally sufficient amounts of β-lactamase (penicillinase) which cleaves the β-lactam ring of the penicillin and nullifies its antimicrobial effect.
- β-Lactamases are produced by certain periodontal pathogens.
- *Note:* If penicillin without β-lactamase inhibitor is prescribed, untreated periodontitis may become exacerbated; multiple periodontal abscesses may develop.

– *Penicillins with β-lactamase inhibitors:*
- For instance, the broad-spectrum penicillin amoxicillin may be combined with the β-lactam ring carrier clavulanic acid (Augmentin), or sulbactam, which have no antimicrobial activity. β-Lactamases have a high affinity with clavulanic acid or sulbactam and are irreversibly blocked.

– *Tetracyclines:*
- Bacteriostatic antibiotics with a broad spectrum of antimicrobial action; interfere with protein synthesis by bacteria.
- High substantivity; systemically administered tetracyclines bind to root surfaces within the pocket and are released even after intake has ceased.
- This may result in about two to four times higher concentrations of tetracycline HCl and doxycycline HCl in gingival exudate than in plasma (Fig. 13.3). Even

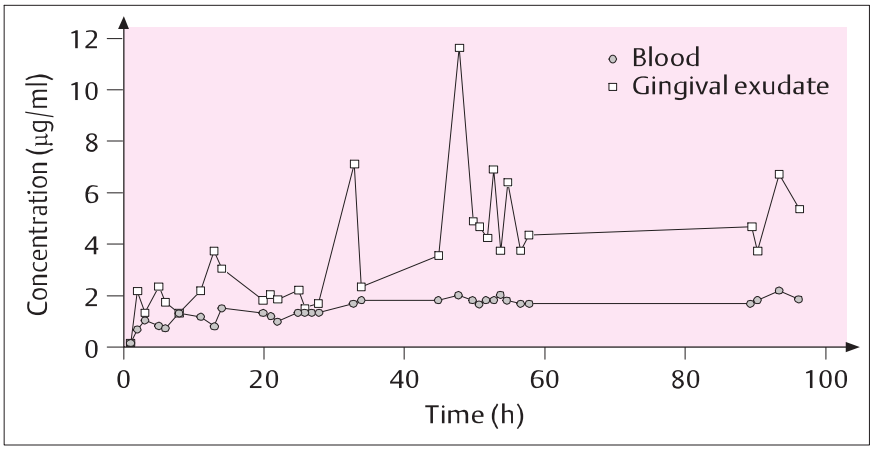


Fig. 13.3 Compared to achievable blood levels, concentrations of tetracycline in gingival exudate may be two to four times higher. Oral dosage: 250 mg every six hours. Note, however, that tetracyclines are insufficiently secreted into saliva. (Adapted from Gordon et al. 1981)

higher concentrations have been reported in gingival exudate for minocycline HCl, which can also be detected in saliva after systemic administration.
  - In contrast, other tetracyclines are rarely secreted into saliva, which might be the main reason for their failure to eradicate oral *A. actinomycetemcomitans*.
  - Irrespective of their antimicrobial effect, tetracyclines inhibit tissue collagenase (see below).
- *Nitroimidazole derivatives* (metronidazole, ornidazole):
  - Interfere with nucleic acid synthesis of bacteria; the exact mode of action is unknown.
  - Small spectrum against protozoa (e.g., trichomonads) and obligate anaerobes.
  - After administration of therapeutic doses, similar concentrations are found in gingival crevicular fluid and serum.
  - Excellent secretion into saliva.
  - Antibiotic of first choice for necrotizing ulcerative gingivitis/periodontitis and refractory infections with obligately anaerobic periodontal pathogens.
  - In laboratory animal experiments, mutagenicity and carcinogenicity have been observed.
  - Cause severe nausea and vomiting if taken in conjunction with alcohol (disulfiram reaction).
- *Clindamycin:*
  - Interferes with bacterial protein synthesis.
  - Is especially active against gram-negative, obligately anaerobic bacteria.
  - May be employed as an adjunct in refractory cases not associated with *E. corrodens* (which is resistant to clindamycin).
  - *Warning:* Pseudomembraneous colitis after superinfection of the gut with *Clostridium difficile*, which is resistant to clindamycin; clindamycine should therefore only be administered if other antibiotics are contraindicated.
- *Azalides*, a new generation of macrolides, in particular azithromycin:
  - Antimicrobial activity against anaerobic and gram-negative bacteria.
  - Reach high concentrations in saliva and gingival tissues.
- *Quinolones*, e.g., ciprofloxacin, ofloxacin:
  - Inhibition of DNA gyrase.
  - Ciprofloxacin has excellent activity against numerous facultatively anaerobic gram-negative bacteria, e.g., nosocomials like *Pseudomonas aeruginosa*.
  - *Note:* Quinolones should not be routinely used in dentistry because of the possibility of resistance development.
- *Combinations:*
  - Metronidazole and amoxicillin have been used together especially in periodontal infections with *A. actinomycetemcomitans*.
  - In patients who are allergic to penicillins, ciprofloxacin may exceptionally be combined with metronidazole.

Local Administration of Antimicrobial Substances

➤ For the use of local antiseptics in mouth rinses and toothpastes see Chapter 10.

➤ Periodontal disease tends to occur locally. Consequently, locally applicable antimicrobial agents may be useful.

➤ In order to be pharmacologically effective, locally applied drugs have to fulfill the following criteria:
- The drug must reach the site of action, i.e., in this case the bottom of the periodontal pocket.
- It must remain there for long enough.
- It must be effective at the concentrations that can be achieved.

➤ Pocket irrigation, preparations with sustained delivery of the drug, and those with controlled delivery have been tested in clinical settings (Fig. 13.**4**):
- *Pocket irrigation* with a blunt canula or special irrigators, e.g., with povidone-iodine (Betadine), or chlorhexidine solution, exhibit only minor effects:
  - The bottom of the pocket is not reliably reached.
  - The time for which the active agent remains in the pocket at effective concentrations is usually too short.
- Preparations with *sustained drug delivery* may exert bactericidal action for extended periods of time:
  - 25 % metronidazole benzoate gel consisting of glyceryl monooleate and sesame

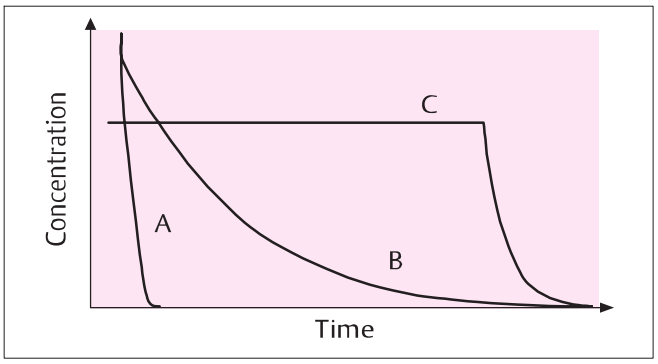


Fig. 13.4 Clearance of intracrevicularly administered antimicrobial drugs.
a Pocket irrigation.
b Drug delivery device with sustained release.
c Controlled delivery of the drug. (Adapted from Tonetti 1997)

oil (Elyzol). The gel is applied with a blunt canula in two sessions one week apart and sets under the influence of gingival exudate/saliva.
  - 2 % minocycline HCl in an ointment containing hydroxymethyl-cellulose, aminoalkyl-methacrylate, triacetin, and glycerin (Dentomycin, Periocline).
- Preparations with *controlled drug delivery:*
  - 25 % tetracycline HCl in a nonresorbable drug delivery device (Actisite). The delivery fiber consists of a plastic copolymer (ethylene and vinyl acetate); it is applied into the pocket with a special plugger and is removed after 7–10 days. The fiber is secured with cyanoacrylate tissue sealant and/or periodontal dressing. Tetracycline concentrations in gingival exudate are higher than 1000 µg/ml for at least one week.
  - Gelatin chip with 34% chlorhexidine (PerioChip). This biologically degradable chip is 5 mm long, 5 mm wide and about 1 mm thick. It is trimmed to fit the pocket morphology. The chlorhexidine concentration in gingival exudate may reach 125 µg/ml for about one week.
  - 8.5 % doxycycline hyclate in a biologically degradable vehicle consisting of poly-DL-lactide and *N*-methyl-2-pyrrolidone (Atridox).

➤ *Critical assessment:* Preparations with controlled drug delivery, especially, can be employed as an alternative to mechanical débridement:
- During supportive periodontal care in case of marked dentin hypersensitivity.
- After considerable loss of tooth substance due to repeat scaling.
- *Note:* Combination therapy (scaling and root planing plus local antimicrobial therapy) had in most cases no additional effect. Possible exceptions include deep periodontal pockets which have not sufficiently responded to mechanical débridement, e. g.:
  - Open furcations
  - Deep intrabony pockets
- However, periodontal pathogens do not only colonize periodontal pockets:
  - Most pathogens are widely distributed within the oral cavity.
  - In cases of bacteria that are difficult to eliminate and may be invasive, such as *A. actinomycetemcomitans*, local antibiotic therapy is not indicated.

➤ *Local antimycotic therapy:*
- Diagnosis of oral candidiasis:
  - White, curd-like patches which leave a red, bleeding surface if removed; or erythematous areas.
  - Cultivation of swabs directly onto an elective medium (Nickerson): assessment after 24–48 hours at room temperature.
  - Cytology of swabs: proof of the mycelium, in some cases hyphae which invade epithelial cells.
  - In chronic cases: biopsy and serology.
- Improvement of oral and, if applicable, denture hygiene. Adjunctive use of chlorhexidine preparations.
- In refractory cases local antimycotic therapy (lozenges, mouth rinsing, chewing gum, gel, ointment, denture varnish) with, for example:
  - Nystatin
  - Amphotericin B
  - Miconazole
- In immune-suppressed patients antimycotic prophylaxis of oral candidiasis is indicated. *Note:* Reinfection of the oral cavity is likely if the gut is not treated simultaneously.

Inhibitors of Tissue Collagenase

- Novel treatment approach as an adjunct to mechanical periodontal therapy. Takes advantage of the inhibitory effect of tetracyclines on matrix metalloproteinases of, in particular, polymorphonuclear granulocytes.
  - Peri- and posttherapeutic, long-term, systemic administration of nonantimicrobial dosages of tetracycline derivatives, e.g., doxycycline hyclate (Periostat): 20 mg/day twice daily for up to 9 months.
  - There may possibly be an increased risk of development of bacterial resistance.
- Presently, tetracycline derivatives are being developed that do not have any antimicrobial action.

Antiinflammatory Drugs

Nonsteroidal Antiinflammatory Drugs

- Metabolites of arachidonic acid are strongly associated with the development of inflammatory periodontal lesions and the modulation and regulation of inflammatory cells:
  - Nonsteroidal antiinflammatory drugs (NSAIDs) interfere with the cyclooxyenase pathway and hence prostaglandin production (see Chapter 3).
  - Prostaglandin inhibitors largely suppress destructive processes during acute inflammation.
  - However, effects on posttherapeutic results after systemically administered NSAIDs such as ibuprofen, naproxen, or flurbiprofen, were found to be rather limited.
  - Routine prescription is not recommended because of frequent adverse side effects:
    - Gastrointestinal complaints
    - Gastric ulcer
    - Impairment of the hematopoietic system
- Infrequently occurring postoperative complaints (pain, swelling) after periodontal surgery may be controlled with paracetamol or ibuprofen after any contraindications have been taken into account:
  - Paracetamol: dosage for adults: 500 mg, 1–4 times a day
  - Ibuprofen: dosage for adults: 200 mg, 1–4 times a day
  - *Warning:* Increased postoperative bleeding tendency after treatment with acetyl salicylic acid (aspirin).

Local Glucocorticosteroids

- Marked antiinflammatory and immunesuppressive effects. Inhibition of early and late inflammatory reactions:
  - Inhibition of vasodilation, edema formation, exudation of neutrophilic granulocytes
  - Inhibition of capillary proliferation, fibroblast proliferation, collagen formation
  - Prevents excessive release of lysosomal enzymes due to membrane stabilization
  - Inhibition of synthesis of proinflammatory cytokines and mediators such as IL-1, TNF-α, PGE_2
  - Inhibiting influence on T cell activity
- Short-term local application of, e.g., prednisolone in case of painful, acute inflammatory bacterial processes:
  - Necrotizing ulcerative gingivitis/periodontitis
  - Pericoronal abscess
- Medium-term local therapy of painful forms of oral lichen planus:
  - 0.1 % betamethasone valerate and 0.5 % vitamin A in a suitable ointment
  - Application three times per day for the first three weeks, then gradual reduction of the dosage over several weeks.

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Index

A

abscesses 51, 58–9, 101
Abt-Letterer-Siwe syndrome 57
acellular afibrillar cementum (AAC) 7–8
acellular extrinsic fiber cementum (AEFC) 2, 4, 7–8
acellular intrinsic fiber cementum (AIFC) 7–8
Actinobacillus actinomycetemcomitans
  bacteriocin 21–3
  biofilms 13, 15
  immunoglobulins 34
  leukotoxin 20, 33
  periodontitis 17–19, 56, 78
  risk 36–7, 172
  tests 80–3
Actinomyces spp. 12–15, 19, 21–2, 79, 114–15
acute phase reactants 29, 84
adhesins 12
adhesion molecules 57
adhesion receptor CD11/18 27
adolescents 41–2, 45
advanced glycated end products (AGEs) 93-4
agranulocytosis 56-7
alcohol abuse 53
alkaline phosphatase 84
allergies 51, 55, 90
alveolar bone proper 2, 6, 9–10
ameloblasts 2, 3, 5
amoxicillin 176
anesthesia 108–9
anorexia nervosa 53
antibiotics 174–9
  prophylaxis 90–1
antibodies 19–20, 32, 36, 85
anticoagulants 91–2
anticonvulsants 52
antiinflammatories 179
antiinflammatory drug, nonsteroidal (NSAIDS) 179
antimicrobials 177
antimycotics 174–9
arachidonic acid 25–6, 107
aspartate aminotransferase (AST) 84
atherosclerosis 92
azalides 177

B

B lymphocytes 29, 31, 33, 36
bacteria 10, 12–13, 53
  culture 78–80
  DNA probes 82
  epidemiology 40
  see also individual species
Bass technique 103–4
Betadine 112
biofilms 13–15
bisecting-angle technique 72–3
bitewing exposures 73
blast cells 2
bleeding 67
  anticoagulants 91–2
  charts 71
  points 121
  probing 169
bone 2
  alveolar proper 6, 9
  files 125, 147
  grafts 134
  loss 86, 165, 171
  wound healing 134
bone fill 131–2
  furcations 150
  tissue regeneration 136
bone morphogenic proteins (BMPs) 135
bradykinin 25
brushing 103–4
  interdental 100, 104–5
  molars 148
  trauma 100
butyrate 24

C

C-reactive protein (CRP) 29
calcium channel blockers 52
calcium phosphate 16
calculus 16, 96, 108
Campylobacter spp. 13, 22, 80
Campylobacter rectus 17–18, 21, 31, 78
Candida spp. 17–18, 50, 53–4
candidiasis 89
Capnocytophaga spp. 13, 15, 21–2, 79
cardiovascular disease 92–3
cathepsin C 57, 85
cellular intrinsic fiber cementum (CIFC) 6–8
cellular mixed stratified cementum (CMSC) 2, 6–8
cementoblasts 2, 4, 7
cementocytes 7–8
cementoenamel junction (CEJ) 3, 21, 142
cementogenesis 3–4, 9–10
cementum 2, 4, 6–9, 32, 143
Charters technique 104
charts 64, 68, 70–1, 76
Chediak-Higashi syndrome 57
chemokines 24, 27, 29–30
chemotaxis 19, 26, 36
children 42, 45, 122
chlorhexidine 23, 106–7, 122, 148
chondroitin sulphate 84
ciprofloxacin 176
citric acid 133
clindamycin 176–7
Cohen syndrome 57
collagen 2, 8, 10
Community Periodontal Index 40
complement 19, 25–8
  C5a receptor 37
computed tomography 74
computer-assisted digital image analysis (CADIA) 74
connective tissue 9, 157
contraceptives 53
corticosteroids 24
Corynebacterium matruchoti 15
Creutzfeldt-Jacob disease 134
crowns 2–3, 167
  Mülhemann's periodontometer 163
  see also dentin, enamel, pulp
curettage 109–16
curettes 110, 116,
  after-five 111
  area-specific 110, 117–8
  flap operations 125
  furcation 125, 143
  gingivectomy 119
  Gracey 111
  intermediates 110
  mini-five 111
  mucogingival 167
  subgingiva1 113
  universal 110, 117, 119
cyclooxygenase 85
cytokines 26–7, 29–30, 32, 36

D

débridement 110, 112
defects 74–6
deformities 51, 59
dendritic cells 29, 32
dental follicles 2
dental lamina 3
dentin 2, 4
  hypersensitivity 108
  predentin 4
dentistry, preventative 47–9
desmosomal fiber bundles 8
desmodontal fiber apparatus 2
diabetes mellitus 19, 93–5, 172
diagnosis 76–87
  clinical 62–71
  defects 74–6
  furcation 66
  medical history 62–3
  radiological 71–4
  tests *see* tests
differentiation factors 134–5
disinfection 106, 116
disorders, hematological 56
distal wedge 129
DNA probes 79, 81–2
Down syndrome 57
doxycycline 95
drugs 174–9
  disease risk 172
  gingivitis 50, 52–3

E

E-selectin 19, 27, 33
ectomesenchymal tissue 2–3
EDTA 133
Ehelers-Danlos syndrome 57
Eikenella corrodens 13, 15, 17–18, 21–2, 78, 80
electrosurgery 123–4
elevators 125, 167
enamel 2–4, 142–3
  matrix proteins 135
endocarditis 89–90
endothelium 27
Enterobacter spp. 18
envelope technique 159
enzyme immunoassay (EIA) 81
epidemiology 38–46
epithelium 2, 4, 6, 19, 34, 133, 136
Epstein-Barr virus 53, 89
erythema exsudivum multiforme 54–5
Escherichia coli 18, 20
Eubacterium spp. 13, 15, 17, 78
Eubacterium nodatum 78
Europe 45
examination, clinical 63–71
Extent and Severity Index (ESI) 41
exudate 13, 67, 84, 169–70

F

Fc receptors 28, 36–7, 85, 172
fibroblast growth factor (FGF) 135
fibroblasts 2, 7, 10, 24, 30–2
files 111, 125, 147
flap operations 124–32, 139, 141
  flaps
    coronally advanced 156–8
    lateral sliding 155–6
    mucoperiosteal ostectomy 148
    papilla preservation 126
    semilunar coronally repositioned 158–9
  Widman technique 127
flossing 104–5
fluorescence, indirect immune 80–1
fluoride 106, 148
follicles 2–4
foreign bodies 55
N-formyl-methionyl-leucyl-phenylalanine (FMLP) 24, 28
receptors 36–7
frenectomy 161–2
fungi 50, 53–4, *see also* individual species
furcations 141–51, 171
  chlorhexidine 148
  curettes 143
  defects 75–6
  diagnosis 66
  fluoride 148
  therapy 143, 149
Fusobacterium spp. 2, 13–15, 18, 80
Fusobacterium nucleatum 17, 58, 78

G

gender 172
genes
  diseases 57
  factors 37
  susceptibility 50
gingivectomy 121–2
gingiva 5–7, 9
  abscesses 58
  attachment 40–1, 43–4, 46–7
  biofilms 13
  bleeding 71
  defense mechanisms 10
  deformities 51, 59
  development 2
  early lesions 30
  electrosurgery 123
  epithelium 6
  exudate 13, 67, 84
  grafts 151–4
  groove 6
  herpes simplex virus 101
  lesions 50–1
  microflora 15–16, 115
  normal 24–5
  plaque 14, 50, 52
  recession 46, 69–70, 114, 154–5
  smoking 96
  sulcus 6
  task of 9
  tetracycline 176–7
Gingival Bleeding Index (GBI) 71
Gingival Index (GI) 39
gingivectomy 119–23
gingivitis 50–5
  adolescents 42
  children 42
  early 29
  established 29–31
  initial 25–9
  necrotizing ulcerative 57–8
  plaque-induced pathogenesis 24–31
gingivoplasty 123–4
gingivostomatitis, herpetic 100–1
γ-globulins 31
glucocorticosteroids 179
glycogen storage disease 57
Gracey curettes 110–11
grafts 134, 151–4, 157
granulocyte-macrophage colony stimulating factor (GM-CSF) 32
granulocytes 32, 36
  neutrophilic 30, 36
granulomas 53, 95
growth factors 134–5

H

Haemophilus spp. 15
Hand-Schüller-Christian disease 57
hemolysin 20
hemophilia 9
Henle-Koch's postulates 17
herpes simplex virus (HSV) 53, 100–1
Hertwig's epithelial root sheath (HER) 2–4, 135, 142
Hill's criteria 17, 38
histamine 25

Howship's lacunae
human cytomegalovirus 53
human immunodeficiency virus (HIV) 19, 36, 89, 100–1
hygiene 71
hyperkeratosis 96
hyperphosphatasia 57

I

immunoassays 81
immunoglobulin A (IgA) 12, 34
immunoglobulin G_2 (IgG_2) 20, 31
immunosuppression 52–3, 172
indices 40, 71
inflammation 25–6, 36, 39–40, 92
  antiinflammatories 179
  cytokines 32
  markers 82
inflammatory cell infiltrates 10
instruments 65–6, 110–12, 116–18, 167
  see also types of instruments
insulin-like growth factor (IGF) 135
integrins 27
intercellular adhesion molecule1 (ICAM-1) 19, 27–8, 33
interferon γ (INF-γ) 32–3, 36
interleukins (IL) 32–3, 36, 84
  IL-1 30, 32–3
    acute phase reactants 29, 84
    disease risk 172
    epithelial cells 27
    monocytes 36
    polymorphisms 85
    susceptibility 37
    tests 84–5
  IL-1β 19, 34, 56
  IL-8
    epithelial cells 24, 28
    macrophages 27, 30
    and *Porphyromonas gingivalis* 19
    receptor 36–7

J

junctional epithelium (JE) 2, 9, 24, 30–1, 34
  interleukin 8, 28

K

Kaposi sarcoma 89
Karlstad study 47–8
Kawasaki disease 89
keratinocytes 32
Klebsiella spp. 18
knives, gingivectomy 121

L

Lactobacillus spp. 12, 15
lamina propria 6, 7
Langer curettes 110
Langerhans cells 29, 57
lasers 123
latex agglutination 81
Leptotrichia buccalis 15
lesions 30–4, 50, 59, 64
  bony 76, 139–40
  periodontal-endodontic 51, 59, 101–2
leukemia 56
leukocidin 20
leukocyte adhesion deficiency syndrome (LAD) 57
leukocyte adhesion receptor CD11/18 27–8
leukocytes *see* polymorphonuclear granulocytes (PMN)
leukotoxin 20, 22, 33, 83
leukotrienes 25–6, 36,
  leukotriene B_4 25, 28, 107
Lewis blood group antigen 27
lichen planus 54
ligament, periodontal 2, 8–10, 136
lipopolysaccharide (LPS) 24, 27, 36, 92
lupus erythematosus 55
lymphocytes 19, 24
  B lymphocytes 29, 31, 33, 36
  T lymphocytes 26, 29–34, 36

M

α_2-macroglobulin 28–9
macrophages 27, 29–34
  hyperactive phenotype 92–3
  hyperresponsiveness trait 19, 35
  IL-8 27, 30
  prostaglandins 27, 29–30, 56
Malassez, epithelial rests of 3, 8
malnutrition 50, 53
mast cells 32
matrix metalloproteinases (MMP) 27, 29, 33, 36, 84
matrix proteins 135
MCP-1 *see* monocyte chemoattractant protein 1
melanosis 96
membranes 136–9, 160
mesenchyme, determinate dental 3
metal salts 106–8
metronidazole 176
Micromonas spp. 13, 15, 17–18, 78
microscopy 15, 78
Miller classification 69, 155–60
mirrors 119, 125, 167
MMP *see* matrix metalloproteinases
Mogibacterium timidum 15
molars 141–52
monocyte chemoattractant protein 1 (MCP-1) 30
monocytes 26, 33
  hyperresponsive phenotype 93
  interleukin 1 36
  prostaglandins 19, 36
mouth 2, 5–6, 9–16
  hygiene 71, 172
  washes 106, 116
mucocutaneous diseases 54–5
mucogingiva 69–71, 151–62
Mülhemann's periodontometer 163

N

Nabers probe 66
natural killer cells (NK) 32
needle holders 125, 167
Neisseria spp. 15, 50, 53
neural crest 2
neutropenia 56–7
NHANES III study 43–4
nitroimidazole 177
Norwegians 46

O

occlusions 59, 162–8
odontoblasts 2–4
odontoplasty 124
Oral Hygiene Index (OHI) 40
orthopantomogram (OPT) 72
osseous defects 75
ostectomy 148
osteoblasts 2, 4, 8
osteoclasts 8, 34
osteopenia 96, 172
osteoplasty 124, 147
osteoporosis 36, 96, 172
oxytalan 2

P

Papilla Bleeding Index (PBI) 39
papillae 2–3
  brushing 100
  loss 100
  preservation 126, 139–40
papilloma virus 53, 89

Papillon-Lefèvre syndrome 57
paralleling technique 72
Pasterella haemolytica 20
pathogens 24–37, 78, 171–2
  see also individual pathogens
patients 12–16, 19, 24–5
pemphigoid 54–5
pemphigus vulgaris 54–5
penicillins 175–6
Peptostreptococcus 13, 15
periocorona 59
periodontal disease
  necrotizing 57–8
  plaque-induced 41–6
Periodontal Disease Index (PDI) 40
Periodontal Index (PI) 40
periodontitis 50–3, 62–88
  adolescents 42, 45
  advanced 35
  aggressive 36, 45, 56, 174
  cardiovascular disease 92
  chronic 56, 175
  and diabetes mellitus 94
  disease transmission 22–3
  early-onset 45
  endodontic lesions 51, 59, 101–2
  epidemiology 38–46
  marginal 31–4
  necrotizing disease 51
  necrotizing ulcerative 57–8
  pathogens 16–20, 36
  and pregnancy 95
  prevention 47–9
  prognosis 87
  progression 34
  smoking 96–7
  susceptibility 19, 85, 172
  and systemic disease 51, 56–7
  teeth 86
  therapy 87–8
periodontium 2, 4–6, 9–11
  abscesses 51, 58–9, 101
  beneficial microorganisms 20–1
  charts 68
  lesions 59
  marginal *see* gingiva
  pockets 21–2
  probing 66–7, 114
  see also alveolar bone proper; cementum; gingiva; ligament, periodontal
phospholipases 25
plaque 13–14, 50, 52
  antimicrobials 106
  charts 71
  control 103–8
  pathogens 24–37, 80
Plaque Control Record (PCR) 40, 71
Plaque Index (PI) 40
plaque-bleeding ratio 170–1
plasma cells 24, 30–1, 34
platelet-derived growth factor (PDGF) 135
pliers 119–21, 125, 145, 167
PMA Index 39
PMN *see* polymorphonuclear granulocytes
pockets 171
  bone fill 131–2
  defects 75
  furcations 150
  periodontal 21–2
  regeneration 141
polishing 111
polymerase chain reaction (PCR) 79, 82–3
polymorphonuclear granulocytes (PMN) 24–6, 28, 30–1, 34, 36
Porphyromonas spp. 13–16
Porphyromonas gingivalis 13, 17–23, 56, 58
  disease risk 172
  immunoglobulins 34
  suppression 33
  tests 78–81
  therapy 114–15
pregnancy 50, 52, 95–6
Prevotella spp. 13–18, 22
Prevotella intermedia 13, 17–18, 52, 58
  tests 78, 80–1
Prevotella melaninogenica 16, 18, 52, 80
Prevotella nigrescens 18, 52, 78, 80
probes 65, 110
  DNA 81–2
  probing 66–7, 114, 169–70
  surgery 65, 100, 119, 125, 167
progenitor cells 132–3
propionate 24
Propionibacterium spp. 15
prostaglandins 24–6, 36
  EU2u 27, 84, 107
    macrophages 27, 29–30, 56
    monocytes 19, 36
prostheses 165–8
proteases 28
α_1-proteinase inhibitor 28–9
Pseudomonas spp. 17–18
puberty 50
pulp 2

Q

Quigley-Hein Index (QHI) 40
quinolones 177

R

race 172
radiography 72–4
radiology 71–4
recession, gingival 46, 69–70, 114, 154–5
regeneration
  furcations 148–51
  tissues 136–41, 159–61
retractors 119, 125, 167
risk factors 36–7, 172
  high-risk groups 49
root complex 142
roots 3, 142–6
  cementum 7–9, 32
  débridement 112
  planing 109–16
  pliers 145
  regeneration 136, 160
  surface 133, 135
Rothia dentocariosa 15
RTX toxins 20

S

sanguinarine 108
scalers 110, 116–17
  scaling 109–16, 160
  surgery 119, 125, 167
scalpels 119–21, 123, 125, 167
Schluger bone file 125, 145, 147
scissors
  flap operations 125
  gingivectomy 119, 121
  mucogingival surgery 167
scrub technique 103
scurvy 53
selectins 27
Selenomonas spp. 15, 31, 78
Sharpey's fibers 2, 8, 133
sialyl-Lewis X 27
smoking 19, 35–6, 96–7
  bone loss 86
  risk 172
socioeconomic factors 172
splints 164–5
Sri Lankans 43, 46
Staphylococcus spp. 17–18, 78
steroid hormones 52
Stillman technique, modified 104
stomatitis, necrotizing 58
Streptococcus spp. 13–15, 22, 50
Streptococcus intermedius 15, 78
Streptococcus mitis 21
Streptococcus mutans 12, 34
Streptococcus salivarius 12
Streptococcus sanguinis 12, 19–20
stress 19, 35, 172
studies, clinical 45–6

subtraction radiography 74
Sugarman bone file 125, 147
sulcus 6
Sulcus Bleeding Index (SBI) 39
supraalveolar fiber apparatus 7
surgery 119
  mucogingival 151–62
  periodontal 119
sutures 125–6, 128, 139–40, 147, 149, 156, 160
  removal 129–30

T

T lymphocytes 26, 29–34, 36
  γδT lymphocytes 30
Tannerella forsythia 15, 17–20, 22, 31
  disease risk 172
  tests 78–9
  therapy 114–15
teeth 2–4, 7–11, 86
  brushing 103–4
  débridement 110
  deformities 51, 59
  extraction 103
  furcation 141–51
  loss 42, 86, 171
  mobility 67, 131, 163, 165–6
  transplantation 10–12
tests 76–8
  bad breath 85–6
  enzymic 79
  genetic 85
  immunological 79
  leukotoxin 83
  markers 82–5
  microbiological 78–82
  molecular 79
  subgingival pathogens 80
  subgingival pockets 79
tetracycline 133, 176–7
therapy, periodontal, risk 169–73
thromboxane 25
tissue, periodontal, regeneration 136–41
tissues
  ectomesenchymal 2–3
  regeneration 130, 136–41, 159–61
    membranes 141
    roots 160
tongue 116
toothpastes 106
transforming growth factor β (TGF-β) 135
transplants
  antibiotics 91
  teeth 10–12
trauma 51, 55, 100–1
  occlusal 59, 162–5
Treponema denticola 13, 15, 17, 19–21
  tests 78–9
  therapy 114–15
Treponema pallidum 20, 50, 53
triclosan 106–7
tumor necrosis factor α (TNF-α) 27, 30, 32–3, 36
  tests 84–5
Turesky modification 40

U

ultrasonic instruments 110

V

Varicella zoster virus 53
vasodilation 25
Veillonella spp. 13–15
viruses *see* individual species
vitamin C 53

W

Widman Technique, modified 126–9
wounds 132–6

X

X-rays 72–4

Tooth Identification

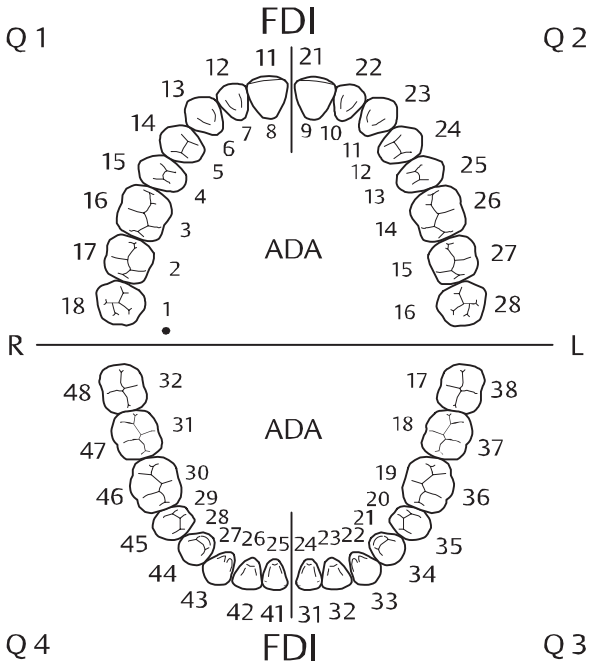
*FDI/International and **ADA Systems

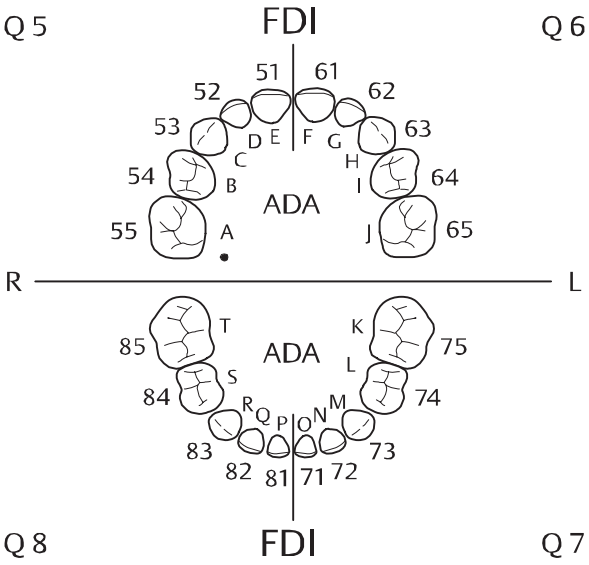
Permanent Dentition

FDI System First digit indicates the quadrants 1–4 Tooth counting from the midline 1–8	ADA System Teeth are numbered counting clockwise from 1–3
Q 1 – upper right	1 (upper right 3rd molar) to
Q 2 – upper left	16 (upper left 3rd molar)
Q 3 – lower left	17 (lower left 3rd molar) to
Q 4 – lower right	32 (lower right 3rd molar)

Deciduous Dentition

FDI System Quadrants count from 5–8	ADA System Teeth have capital letters A–T
Q 5 – upper right	A (upper right 2nd molar) to
Q 6 – upper left	J (upper left 2nd molar)
Q 7 – lower left	K (lower left 2nd molar) to
Q 8 – lower right	T (lower right 2nd molar)





*FDI Fédération Dentaire Internationale (International)
**ADA American Dental Association (USA)